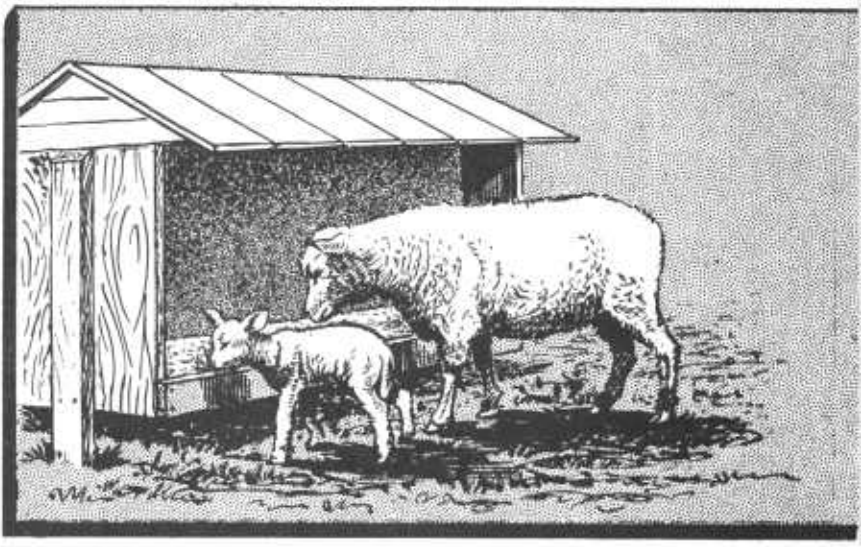
# Parasites and Parasitic Diseases of Sheep



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# CONTENTS




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# Parasites and Parasitic Diseases of Sheep

By J. T. LUCKER and A. O. FOSTER, *parasitologists, Animal Disease and Parasite Research Division, Agricultural Research Service*[1]

SHEEP are very susceptible to attacks by parasites, and probably suffer more severely from such attacks than any other kind of livestock. The importance of parasites and parasitic diseases of sheep is the more evident because sheep are much less subject to serious bacterial or virus diseases than other kinds of livestock. The steady loss of sheep, mutton, and wool from disease is due mostly to parasites.

Damage to sheep from parasites is greatest as a rule among lambs and yearlings. It is fairly well established that in general young animals are more easily infected by parasites than older ones. Sometimes, however, the resistance of very old sheep breaks down, and they then become very susceptible to infection.

The sheepman should make every possible effort to prevent his sheep from becoming infested with parasites. Giving medicine to animals that have become diseased is a last resort. He can go far toward protecting his flock from infestations through such practices as good feeding, proper stocking, sanitation, and special care of breeder ewes and new lambs. In addition, he usually needs to take special measures against individual species of parasites. To do this most successfully he must have a thorough knowledge of how the various species attack sheep and of their life histories.

Many of the most injurious internal parasites of sheep live in the animals' digestive organs. Eggs of these parasites pass out with the manure of infested sheep and as a result pastures become contaminated. To prevent infestations from spreading in this way, sheepmen rotate flocks among different pastures, feed nursing lambs in bare lots, feed grown sheep from racks or board floors, and give rest periods to pastures on which sheep have been grazed. Because other kinds of livestock are not readily infected with sheep parasites, they are often rotated with sheep. Likewise breeder ewes, because they have greater resistance to some parasites than lambs, are sometimes grazed on sheep pasture where lambs will be grazed later.

Some parasites, such as mange mites and lice, pass directly from sheep to sheep; others, such as liver flukes and certain tapeworms, infest sheep in the adult stage but must pass through certain immature stages in other animals. (In this connection the other animals are called intermediate hosts. The life history of a parasite is said to be in-

[1] This is a revision of an earlier edition by M. C. Hall, G. Dikmans, and W. H. Wright. Valuable assistance in revising the bulletin was given to the authors by G. Dikmans, formerly parasitologist, Bureau of Animal Industry, and K. C. Kates and F. D. Enzie, parasitologists, Animal Disease and Parasite Research Branch, Agricultural Research Service.

direct if there is an intermediate host.) Also, some tapeworms of dogs pass through their immature stages as bladderworms in the muscles and in the viscera or the visceral cavity of sheep. Dogs become infested with the tapeworms as a result of feeding on uncooked sheep meat or viscera containing bladderworms. The tapeworm eggs, when taken into the digestive system of sheep, develop into bladderworms. For these reasons, farm dogs should be kept free of parasites and stray dogs should not be permitted to wander over sheep pastures.

Many parasites are likely to infect sheep that are allowed to graze on low, wet pastures. Animals infected under such circumstances should be separated from the flock, and low, wet parts of pastures should be drained, fenced, or filled in.

While making every effort to prevent infection with parasites, the sheepman should be alert to detect early symptoms of parasitic diseases in his flock, and needed treatment should be given promptly. A flock free of such diseases is rare, for many kinds of parasites are always present on most sheep farms. By the time a sheep (particularly, a lamb) has become heavily infested with parasites, it has usually suffered permanent injury. Certainly, it has taken part in contaminating the pasture on which it and other animals are grazing.

If sheep are losing condition, are becoming thin, and either have diarrhea or are constipated, but have little or no fever, the sheepman should suspect that parasites are causing the trouble. Blood-sucking parasites produce anemia; that is, they cause the blood of a sheep to become thin and pale. Often together with anemia there is edema, or dropsy. In this condition, fluid accumulates in the pendent parts (underside) of the sheep's body.

When sheep become diseased, it is advisable to consult a veterinarian. Errors in diagnosis made by unskilled persons waste valuable time and lead to useless or even injurious treatment. Drugs used to kill parasites are commonly poisonous substances that can do much damage if wrongly applied.

If you see that your sheep are becoming unthrifty, act at once to find out what is the trouble. Do not wait until one of them dies, but sacrifice one of the worst affected. A post mortem examination of one animal may reveal the nature of the ailment and save the others. In particular, if you wait until disease has reached an advanced stage in lambs, they may become so poor and weak that they cannot survive treatment or, if they do survive, will in most cases be stunted and unprofitable.

## EXTERNAL PARASITES [2]

External parasites are those that live on the skin surface, in the layers of the skin, or at the roots of hairs. Most of the external parasites of sheep, such as screw-worms, fleece worms, sheep ticks, and lice, are insects, which have 6 legs in the adult stage; others, such as the mites and true ticks, have 8 legs (or appendages that look like legs) in the adult stage. Both these classes belong to the group called arthropods. Some of the external parasites, namely scab mites, lice, and sheep ticks, spend their whole lives on sheep. Others attack sheep only occasionally, or are parasites only during certain stages of their development.

### Lice [3]

**Location.**—The lice that infest sheep live on the skin. They crawl about on the wool fibers or hairs

[2] Scientific names of parasites that are referred to in the text by their common names only are listed at the end of the bulletin.

[3] For additional information see Leaflet 308, Control of Lice and Sheep Ticks on Sheep and Goats.

and cling to them while feeding. The sucking body louse is commonly found in colonies on various parts of the body, including the face. The foot louse, which is likewise a sucking louse, usually lives on the lower parts of the legs, below the true wool. The biting louse lives on various parts of the body.

**Appearance.**—The lice are yellowish brown.

The sucking body louse has a head somewhat longer than its thorax. The male is about one-twelfth inch in length, the female about one-tenth inch. Usually the wool in the region where this louse has attacked is discolored and contains numerous brown particles, the excretions of the louse.

The foot louse (fig. 1, *A*) has a short head, as wide as it is long, merging into the thorax, with reddish oblique bands on each side. The female is about one-twelfth inch long and one-twenty-fifth inch wide; the male is broader and flatter.

The biting louse (fig. 1, *B*) has a head about as wide as long, with a broad, round forward end. The male is about one-twentieth inch long; the female, slightly longer.

Lice are readily found by carefully examining infested animals, particularly in direct sunlight.

**Life history.**—The eggs of sheep lice are attached to the hair or wool on the parts customarily infested by the adult lice. The eggs of the sucking lice hatch in 10 to 18 days; those of the biting louse, in 5 to 8 days ordinarily and in 10 days in cold weather. The young lice become mature, and the females begin laying eggs, about 2 weeks after hatching.

**Distribution.**—The biting louse is rather common in the United States. The sucking body louse is fairly common on sheep in the Southwest. The foot louse has been found on sheep in various parts of the country.

**Damage and symptoms.**—Sucking lice suck blood. Biting lice feed on the outer layer of the skin and material on the surface of the skin. Lice usually cause little trouble in summer but become more numerous and annoying in winter. Both biting and sucking lice cause itching and irritation. This interferes with feeding and nutrition, and prevents animals from fattening or keeping in condition as they should.

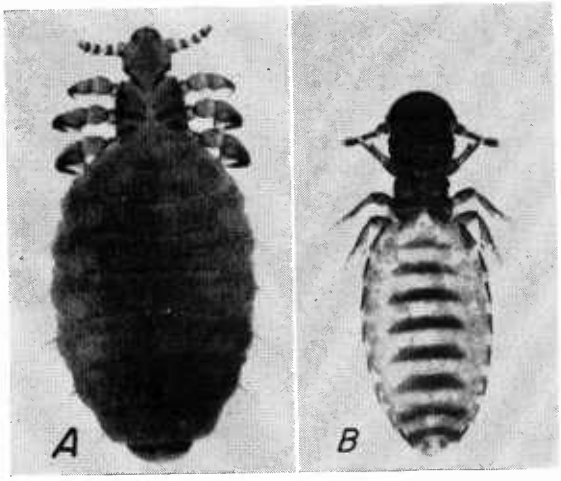


FIGURE 1.—*A*, Sheep foot louse, female; *B*, sheep biting louse, female. (Each about 30 times natural size.)

Also, the itching leads to scratching, which results in loss of wool and may result in cuts and bruises. The loss of nervous energy and interference with feeding caused by lice tend to stunt the growth of young animals and predispose them to various diseases by lowering their vitality. Where numerous biting lice cluster, they cause actual sores. Sucking lice, if numerous, extract considerable blood and lymph. Finally, the excretions of the lice soil the wool; this is particularly true of the sucking body louse.

**Treatment and control.**—Dipping is the one reliable method of eradicating lice from sheep and lambs. The most efficient insecticides are rotenone, DDT, TDE, methoxychlor, BHC (benzene hexachloride), lindane, toxaphene, and chlordane. These have almost completely replaced older louse-killing materials such as nicotine, sulfur, coal-tar creosote oil, cresylic acids, and arsenicals. The newer treatments, if properly used, can be relied upon to eradicate lice in a single dipping; with the older treatments, two dippings at an interval of 14 to 16 days were almost always required.

The dilute rotenone dip recommended for sheep ticks (p. 5) kills a large percentage of the lice, but one containing 1 pound of derris or cube powder in 100 gallons of water is more reliable. Aqueous suspensions or emulsions of DDT at concentrations of 0.20 to 0.25 percent are effective and safe. For eradication of the foot louse, a 0.5-percent DDT dip is the best treatment known. To make a 0.25-percent DDT dip, either 4 pounds of wettable 50-percent DDT powder or 1 gallon of a 25-percent DDT emulsifiable concentrate should be thoroughly mixed into 100 gallons of water.

TDE, methoxychlor, toxaphene, and chlordane should be used in the same strengths as DDT. The concentration in which the gamma BHC, or lindane, is commonly used against lice is 0.06 percent, the same one recommended for eradicating the common scab mite; however, half this concentration has often proved effective against lice. To prepare a BHC dip containing 0.06 percent of the gamma isomer (the active ingredient), use 8 pounds of wettable BHC powder that contains 6 percent of the gamma isomer for each 100 gallons of water.

Inasmuch as commercial products vary in content of the active ingredient, it is advisable to follow the directions on the package. In any case, the products should be used in strengths about the same as those mentioned above.

Where sheep are infested with the biting louse only, sodium fluoride may be applied in the form of a powder to get rid of them. A single application suffices. Rub the powder into the skin at several places, or apply it with a dust gun. It is of no value against sucking lice. Sodium fluoride should not be applied to mucous membranes, such as those of the mouth and anus.

To prevent clean animals from becoming infested with lice, do not allow them to come into contact with lousy ones and keep them out of sheds, pens, enclosures, and pastures where lousy stock have been within 15 days. If sheep are treated with DDT or with one of the newer chlorinated hydrocarbon insecticides, the insecticide remains active in their wool over a long period. Thus if lice accidentally left alive in sheds or lots get on the treated animals, the insecticide in the wool kills them.

## Sheep Tick[4]

**Location.**—The sheep tick, known also as the ked, lives in the

[4] For additional information see Farmers' Bulletin 2057, The Sheep Tick and Its Eradication.

wool and on the skin. Its favorite locations are the neck, breast, shoulders, belly, and thighs.

**Appearance.**—This parasite is not a true tick but a wingless fly (fig. 2). It has 6 legs, whereas a full-grown true tick has 8. The mouth parts are very similar to those of other flies. Sheep ticks are reddish or gray brown and are about one-quarter inch long; therefore, they can easily be distinguished from lice. They are distinctly divided into head, thorax, and abdomen. This characteristic helps to distinguish them from true ticks.

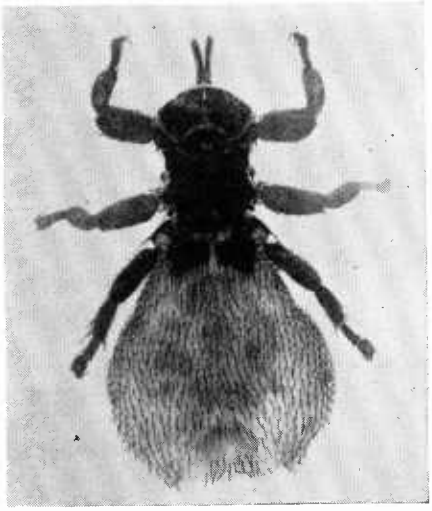

FIGURE 2.—Sheep tick, engorged female. (Much enlarged.)

**Life history.**—The young brought forth by the sheep tick are commonly called "eggs," but each of them actually is an early pupa, a much more advanced stage of development than the egg. The pupa is deposited by the female in the wool and is attached to the wool by a gluelike substance. When deposited it is covered with a soft white membrane, which in about 12 hours becomes a hard brown case. The young tick emerges from the pupal case in about 19 days in warm weather, or in about 24 days in cold weather. The tick is almost full grown when it emerges, and it becomes mature 3 to 4 days later. After mating, the female may deposit its first pupa in 8 to 10 days.

**Distribution.**—The sheep tick is widely distributed and is common on both farms and ranges. It is more common on coarse-wool and medium-wool sheep than on sheep having fine wool.

**Damage and symptoms.**—The sheep tick is a bloodsucker, causing loss of blood, great irritation, interference with feeding, and consequently poor growth and reduced vitality. In addition to causing wool deterioration by injuring the sheep's health, it lowers the value of the wool by soiling it with excretions and pupal cases. When sheep bite, scratch, or rub and their fleece becomes ragged, the sheepman should examine them for sheep ticks (or other parasites). Sheep ticks, if present, can be found by parting the wool.

**Treatment and control.**—Dipping is the best method of applying insecticides to eradicate the sheep tick. Pressure spraying and power dusting have been used with reasonable success. The so-called dilute rotenone dip is effective and economical. It is prepared by mixing 8 ounces of fine derris or cube powder, containing 5 percent of rotenone, into 100 gallons of water. The other dips recommended for the eradication of lice (p. 4) are effective also against the sheep tick. Sprays may be prepared with the same materials used for dips; they should usually have about twice the strength of the dips. For power dusting, the sheepman is advised to use derris or cube powder, containing 5 percent of rotenone, mixed with a suitable inert carrier at the rate of 1 pound of insecticide to 9 pounds of the carrier. Special chute and nozzle arrangements are necessary for dusting rapidly and successfully.

For best control of sheep ticks, infested flocks should be dipped

about 10 days after shearing. Lambs should be included. Shearing removes most of the ticks from the shorn animal, but after ewes are shorn the number of ticks on the lambs may increase rapidly. If sheep are treated with DDT or some other chlorinated hydrocarbon insecticide, the insecticide remains active in their wool and protects them from sheep ticks over a long period.

## Sheep-Scab and Itch Mites[5]

### COMMON SCAB MITE

**Location.**—The common scab mite lives in colonies on the surface of the skin, usually on the withers, back, sides, and rump.

**Appearance.**—The adults (fig. 3) are whitish, oval-bodied, 8-legged, and very small. The female is only about 1/40 inch long, and the male is slightly smaller. However, they can be seen with the naked eye, especially if skin scrapings containing them are placed on a dark background. If warmed by sunlight or otherwise, they may move about and so be more easily seen.

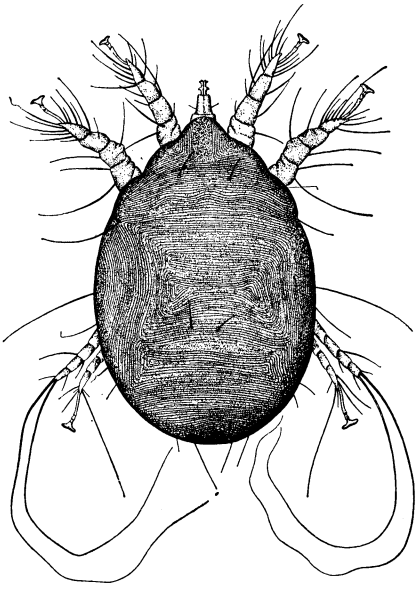

FIGURE 3.—Common scab mite. Female, as seen from above. (Greatly enlarged.)

[5] For additional information see Farmers' Bulletin 713, Sheep Scab.

**Life history.**—The female usually deposits about 15 to 24 eggs in the course of her life and may deposit 90. The eggs usually hatch in about 3 to 4 days. The 6-legged mites that emerge from them develop into 8-legged adults within 7 to 8 days. After mating, the females begin depositing eggs within 3 or 4 days.

**Distribution.**—Common scab, the disease caused by this mite, once was widely distributed over the United States and was the greatest pest that sheepmen had to contend with. Now it has almost been eliminated, although it is still prevalent in scattered areas. Complete eradication is the goal. Individual owners should promptly report cases of scab or cases that they think may be scab to local livestock sanitary authorities.

**Damage and symptoms.**—The mites prick the skin and suck the blood serum. A group of punctures becomes surrounded by a reddish inflamed area. Serum from the tiny wounds, and pus from swellings that develop around them and rupture, form the scabs from which the disease takes its name (fig. 4). The bites of the mites cause itching, and this leads to scratching, rubbing, and biting, which add to the inflammation and cause some bleeding as the scabs are torn off and sores form. The sores afford favorable conditions for bacteria and become infected. The skin reacts to the continued inflammation by becoming thickened and wrinkled.

The first symptoms, as a rule, are rubbing or scratching. These actions roughen and break the wool. Also, the animals bite the affected spots to relieve the itching. Tufts of the wool are torn out and are seen hanging from the fleece. Finally the wool disappears from large

FIGURE 4.—Common scab in an early stage.

areas, leaving the scaly, crusted, grayish diseased skin bare (fig. 5). Affected sheep become restless. They waste considerable energy and time rubbing, scratching, and biting themselves. Loss of time and energy from feeding results in loss of condition. Many sheep will die of scab unless treated. Sheep with advanced scab are so weakened that they readily fall victims to other diseases.

Symptoms like those of scab may be caused by lice, sheep ticks, true ticks, bearded seeds, cactus spines, eczema, wildfire, summer sores, rain rot, shear cuts, sunburn, inflammation of the sebaceous glands, or alkali dust. However, whenever the sheepman observes itching, loss of wool, or other symptoms that have been mentioned as those of scab he should always suspect scab and take steps to get a definite diagnosis. Scab is too serious a disease to warrant taking any chances on its spread.

**Treatment and control.**—The one satisfactory treatment for scab is dipping. As soon as possible after scab is detected, dip all animals of an infected or exposed flock at one time. A 0.06-percent suspension of gamma BHC, or lindane, prepared with wettable powder, is effective in a single application. Commercial BHC costs less than lindane and is equally satisfactory. Eight pounds of wettable BHC that contains 6 percent of the

FIGURE 5.—An advanced case of common scab.

gamma isomer (the active ingredient) is sufficient for 100 gallons of dip. Products containing the gamma isomer in other percentages should be used in amounts giving equally strong dips. The lime-sulfur dip and the nicotine dip are effective if used twice with an interval of 10 to 14 days between dippings. The 10-day interval is preferable.

It cannot be emphasized too strongly that no dipping program for eradicating scab will be successful unless (1) the dip contains the correct amount of insecticide, (2) every sheep is dipped, and (3) every sheep is held in the vat for at least 2 minutes. The BHC and lindane dips retain their power on the skin and in the wool long enough to kill all mites that hatch from eggs remaining fertile after the dip is applied. In general, therefore, if they are applied as directed they completely eradicate the scab mite. The lime-sulfur and nicotine dips are effective up to 60 days, if not washed out by rain or otherwise. It is advisable, when these older dips are used, not to return dipped sheep to pasture that has been occupied by scabby sheep within 30 days. After dipping for scab eradication, avoid adding to the flock new sheep that may be infected.

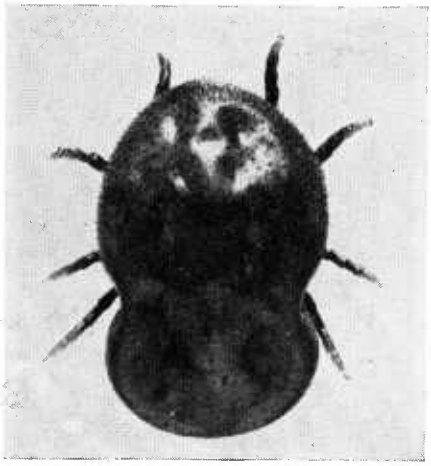

FIGURE 6.—Engorged nymph of the spinose ear tick. (About 5 times natural size.)

### OTHER SCAB AND ITCH MITES

Four other mites affect sheep: The foot-scab mite, the head-mange mite, the follicular-mange mite, and the sheep-itch mite. The treatments used for common scab are effective against foot scab, also. Head mange is harder to cure, but it usually yields if one of the dips used for common scab is applied to the head repeatedly. No reliable treatment for follicular mange is known. The sheep-itch mite apparently is destroyed by the lime-sulfur dip, but it does not yield to the BHC or the lindane dip.

## Other External Parasites

Some species of true ticks infest sheep in the United States occasionally. True ticks have the thorax and abdomen fused, with the head not conspicuously separate. The one that attacks sheep most often is the spinose ear tick,[6] which is most prevalent in the Southwest. Adults of this species live on the ground and deposit their eggs there. A 6-legged seed tick emerges from the egg. When a seed tick gets onto a sheep it crawls into the outer canal of the animal's ear. There it attaches itself well below the hairline, sucks blood from the tender skin, and becomes an engorged, grublike larva. The larva molts to form an 8-legged nymph, which is covered with numerous small spines. After spending months in the ear the blood-engorged nymph (fig. 6) crawls out, drops to the ground, conceals itself in a dry protected place, and transforms into an adult tick.

The ear canals of infested animals often become plugged with wax and

[6] For additional information see Farmers' Bulletin 980, The Spinose Ear Tick.

the excretions of the ticks. Animals thus affected shake their heads or turn them from side to side. Infestations may cause serious injury. Occasionally, they cause death.

Several species of blowflies attack sheep. One, the screw-worm fly, is especially prevalent in the Southwest and South. Its maggots, known as screw-worms,[7] cannot eat through the unbroken skin of a healthy sheep, but they can enter the flesh through any kind of break in the skin. Operations such as castration and dehorning, skin diseases (especially around the natural body openings), tick bites, scratches, and fresh cuts caused by shearing can lead to infestation.

The screw-worm fly (fig. 7) is bluish green, with three dark stripes along its back and an orange or reddish face. The female lays her eggs in masses on the edges of a wound or on a nearby spot of blood. They hatch in 6 to 12 hours. The small maggot (fig. 8) that issues from each egg promptly burrows into the wound, and rapidly eats into the live flesh. After 3 to 10 days, the maggot has reached full growth and is about two-thirds inch long; it then leaves the wound, drops to the ground, burrows into the soil, and transforms into a fly—within 7 to 14 days, in warm weather.

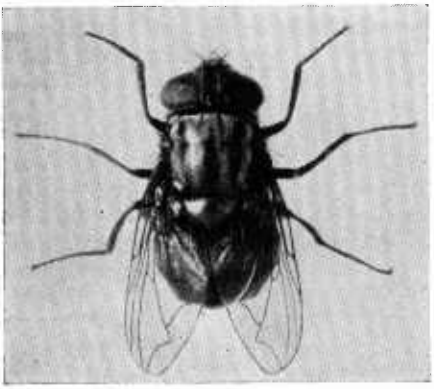

FIGURE 7.—Female screw-worm fly, as seen from above. (Much enlarged.)

[7] For additional information on screw-worms, write to the Entomology Research Division, Agricultural Research Service, Washington 25, D. C.

The maggots cause bleeding and pain as they penetrate into the flesh. By their feeding, each of them forms a small pocket in it. As they enlarge the wound, it attracts more flies. Finally, vital organs may be exposed. This, or absorption of poisons from the extensive wound or wounds, may kill the sheep.

In regions where the screw-worm fly occurs, it usually is the first to attack sheep that have skin or flesh wounds. Two other blowflies, the secondary screw-worm fly and the fleeceworm fly, frequently attack such sheep, especially those recently dehorned or castrated. These flies,

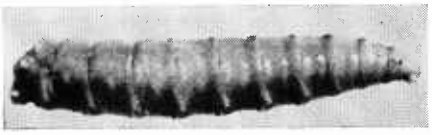

FIGURE 8.—Screw-worm maggot, side view. (Much enlarged.)

also, lay their eggs near the wound, usually on its edges, and their maggots move into it to feed. As a rule, the maggots feed on the already damaged tissues, but they enlarge the wound and cause serious damage.

Blowflies also attack sheep whose wool is soiled and wet with urine and manure. The fleeceworm fly and certain species of the genus *Phaenicia* [=*Lucilia*] often attack such sheep. Less frequently, the secondary screw-worm fly does so. The larvae that hatch from eggs that any of these blowflies deposit in the fleece are called fleeceworms or sheep-wool maggots. They spread over the skin, feed on it, and severely irritate it. The infestation soon causes sheep to become feverish and weak and may kill them.

The screw-worm fly breeds exclusively in the living flesh of warm-

blooded animals. Aside from it, these blowflies ordinarily breed in carrion. They produce large broods and in certain areas sometimes become very abundant. The secondary screw-worm fly has about the same distribution as the primary. The other species are widely distributed, but infest sheep principally in the Southwest, the South, and the Pacific Northwest.

**Treatment and control.**—The best treatment for infestation with ear ticks is 5 percent of chlordane in pure pine oil applied with a spring-bottom oiler having a rubber-tipped spout. Lindane, toxaphene, BHC, and some other chemicals also are effective.

To safeguard his flock against screw-worms, the sheepman should do all he can to prevent unnecessary wounds and should treat wounds promptly. If wounds become infested with screw-worms, preferably he should treat them with a special formulation containing 3 percent of lindane, known as EQ 335 screw-worm remedy. Other effective formulations are EQ smear 62 and EQ smear 82, which contain benzene and diphenylamine.

Infestation with fleeceworms can often be prevented by clipping the wool from the crotch region early in the spring and by guarding against conditions, such as infestation with parasites of the digestive tract, that cause soiling of the wool in this region. The chances of infestation are reduced by early lambing, early shearing, and prompt burning or burial of carcasses.

For treating wounds infested with fleeceworms, formulations containing 0.3 percent of lindane are recommended. The best of these is EQ 335 screw-worm remedy diluted with 9 parts of water. Other screw-worm remedies give good results. Water emulsions or suspensions of chlordane and toxaphene, prepared according to manufacturer's instructions, are useful.

## INTERNAL PARASITES

Internal parasites are those that live inside the body tissues or cavities of the host animal. The internal parasites that infest sheep include arthropods, roundworms, flukes, tapeworms, and protozoa. Only the more important ones are discussed here.

### Arthropods

Although most of the arthropods that attack sheep are external parasites, a few go through some of their life stages as internal parasites of sheep; the most important of these, and the only one discussed here, is a fly whose larvae live in the head and are called head grubs. This fly is called the head maggot fly, sheep nasal fly, sheep gadfly, or sheep bot-fly. Infestation with its larvae is known to sheepmen as grub-in-the-head.

#### HEAD GRUB

**Location.**—The grubs (larvae, bots, or maggots) occur in the nostrils of sheep, their frontal sinuses (in the forehead), and their maxillary sinuses (in the upper jawbone).

**Appearance.**—The youngest grubs are creamy white and less than one-twelfth inch long. A grub that has become fully grown in the sheep's head (fig. 9) is usually over four-fifths inch long and about one-third inch wide. Its body is divided into 11 segments, each having a rather flat, spiny lower surface and an arched, smooth upper surface. At the head end are 2 large hooks, and at the tail end are 2 rounded breathing pores. Commonly there are about 10 crosswise bands of a dark color, often brown, on the upper

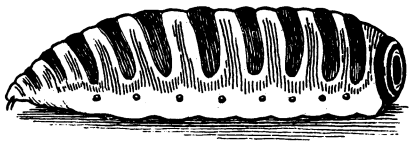

FIGURE 9.—Full-grown head grub from sheep. (Enlarged.)

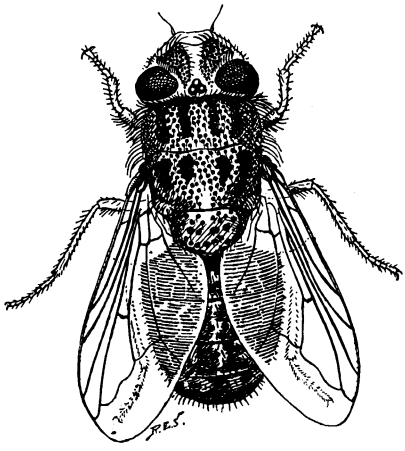

FIGURE 10.—Adult fly, called the head maggot fly, that causes grub-in-the-head. (Much enlarged.)

part of the body and the rest of the body is yellowish or grayish.

**Life history.**—The adult fly (fig. 10), which looks something like an overgrown housefly, is most active during the summer, usually in June and July, but in regions having a long warm season it may attack sheep from late spring to early fall or in all but the coldest months. Usually it is quiet in the early morning and late afternoon and attacks during the heat of the day.

The female fly deposits a tiny grub on the edge of the nostril. The grub enters the nostril, gradually moves up the nasal passage, and usually makes its way to a sinus. In this cavity it feeds and grows until it reaches maturity and is ready to leave the sheep. If the grub enters the nostril in the hot season, it may reach maturity in the sheep's head about a month later. In regions having cold winters, grubs that get into the nostrils in the fall apparently remain in the nasal passages over winter, move to the sinuses the following spring or summer, and then grow to maturity. Where the cold season is long and the fly season is brief some grubs may not reach maturity in less than about 10 months. At maturity the grubs return to the nasal passages, fall to the ground, burrow into the soil, and become pupae, with hard and leathery skin. The pupae lie inactive for from 3 weeks to 2 months, according to soil temperature and moisture. At the end of this period the adult flies emerge from the pupal cases, crawl to the soil surface, and take wing. Under very favorable conditions, the entire life cycle may be completed in 2 to 3 months. Usually it takes longer.

Grubs that die in the sinuses, sometimes because they have become too large to get out, usually harden to a stonelike condition.

Where cold weather kills both the adult flies and the pupae, the parasite's survival apparently depends on the overwintering of some young grubs in the nasal passages of sheep.

**Distribution.**—This parasite appears to be prevalent throughout the United States wherever sheep are kept.

**Damage and symptoms.**—As the grubs crawl about in the nostrils, they cause irritation, which results in inflammation. This causes the mucous membranes to produce a great deal of secretion and to become thickened. If bacterial infection sets in, the secretions become very thick. These conditions interfere with breathing and tend to impair the animal's general health. The sinuses may become filled with pus, and poisonous substances associated with it may be absorbed.

Sheep often run when the fly attacks them. They stop feeding, become restless or even frantic, and press their noses against the ground or against other sheep. The characteristic symptom of grub-in-the-head is a profuse nasal discharge resembling that caused by a head cold. The discharge is thin at first, but it soon becomes thick and discolored. In addition to having difficulty in breathing, the sheep may sneeze frequently. The eyes

become inflamed. The head may be carried low and moved about in a peculiar way, as if in an effort to get rid of an obstruction. The sheep may lose condition because they lack appetite or are so distracted that they cannot graze enough. In severe cases, they may have convulsions and die.

Post mortem examination may reveal rows of black dots on the membranes of the nostrils and sinuses, due to hemorrhages from punctures caused by the grubs. If only live grubs are present, the main evidence of damage may be an excessive amount of secretion. If dead grubs are present, the lining of the sinuses is likely to be inflamed, discolored, and thickened and the sinuses may contain quantities of thick, discolored secretion.

**Treatment and control.**—An effective treatment for grub-in-the-head is thorough, careful irrigation of the nasal passages with a 3-percent solution of saponated cresol. About 1 fluid ounce of the solution is injected into each nostril, under a pressure of from 35 to 45 pounds, from a suitable pressure tank. A preparation containing 15 percent of tetrachlorethylene and 85 percent of mineral oil emulsion is reported to have been used in a similar manner with success. In areas where the winters are cold, the best time to treat is in the fall or early winter, as soon as possible after the botflies have been destroyed by freezing temperatures. Where the winters are warm or moderate, treatment has to be applied at short intervals, because it is primarily destructive to the very young grubs. Other treatments are reported to be of some value, but they require further investigation.

### Roundworms

The roundworms (nematodes) that infest sheep are cylindrical and somewhat resemble short hairs or threads. The body wall is usually rather transparent; sometimes the tubular internal organs can be seen through it. The sexes are usually separate. Males are usually smaller than females.

The females produce eggs. The eggs of a few species develop and hatch while in the sheep, or even while in the worm, but those of most species hatch in the open. When an egg hatches, a microscopic larva emerges from it.

Most of the roundworm species infesting sheep live in the digestive tract or lungs, and their eggs or young larvae are carried out with the sheep's manure. Many of these species have a direct life history. After being carried out, their eggs or young larvae develop in the open to a stage in which they are infectious to sheep. This process, because it takes place in the open, is called free-living development. The infective larvae of a few species can penetrate into a sheep's body through the skin. Those of most species, and infective eggs, pass into the sheep's body only when swallowed by the animal together with forage. After the infective egg or larva gets into the sheep, it gives rise to an adult worm. When the worm matures, the cycle of development starts again.

Other roundworms that infest the digestive tract or lungs of sheep have indirect life histories. The eggs or young larvae of these species cannot develop in the open into the larval stage that is infectious to sheep; they can develop to that stage only within an intermediate host, such as a beetle or snail that feeds on, or comes in contact with, the manure containing them. Sheep become infected with these species by swallowing the intermediate hosts.

Larvae that infect a sheep by penetrating its skin migrate through the body and are carried by the blood to the intestine or to another special location where they can develop into adult worms. Some

of the species of larvae that infect sheep when swallowed also migrate through organs or tissues before they reach, or finally settle down in, the locations where they can develop into adults. Such migrations may cause considerable damage to parts of the body other than the one where the species matures.

### GULLET WORM

**Location.**—The gullet worm usually is found "sewn" into the lining of the gullet. Occasionally it is seen in the cavity of the paunch (the first stomach).

**Appearance.**—When an infested gullet is held up to a strong light, the wavy, snakelike outlines of the worms are seen rather easily. When removed from the lining membrane, the worms are seen to be whitish, very long, and very slender (fig. 11, *I*). The females usually are about 3 to 4 inches long, the males about 1½ inches; their diameter is about equal to that of a coarse thread.

**Life history.**—Indirect. Several kinds of dung beetles serve as intermediate hosts. When eggs that have passed out with the droppings of infested sheep, cattle, swine, and wild ruminants (such as deer) are eaten by one of these beetles, the larvae that emerge from the eggs pass from the gut to the body cavity of the beetle and there develop to the stage that can infect sheep. When infested beetles are eaten by a sheep, these larvae develop into adult worms in its gullet.

**Distribution.**—This parasite is widely distributed. Infestations are fairly common but usually are not heavy.

**Damage and symptoms.**—Gullet worms are not known to affect the health of sheep, but they produce winding channels in the membrane lining the gullet as they move through it, and weasands containing them are not fit to eat.

**Treatment and control.**—No methods have been developed for treatment or control of gullet worm infections.

### LARGE STOMACH WORM

**Location.**—The large stomach worm, also called the twisted or common stomach worm, is a parasite of the abomasum, or fourth stomach.

**Appearance.**—The worms (fig. 11, *B*) are from ¾ inch to 1½ inches long and about as thick as an ordinary pin. The females are larger than the males and appear to be marked with alternating spiral, or twisted, reddish and whitish bands. In the rear half of the body of the female a projecting flap may be seen on close examination. The male is rather uniformly pinkish. Its tail end is flattened and widened.

**Life history.**—Direct. The eggs, which pass out in the manure, develop and hatch in about 24 hours under the most favorable conditions of warmth and moisture. The young larva that emerges from the egg develops and transforms into an infective larva in about 4 days under these conditions. Extreme dryness, or freezing or subfreezing cold, kills the eggs and young larvae in a few days. Even when moisture is ample the eggs and young larvae die, after a longer time, if the prevailing temperatures, although above freezing, are not high enough to permit their development. Within the range of temperatures that permit it, their development into infective larvae proceeds more slowly the lower the temperature.

The infective larva is enclosed in, and protected by, a rather thick, impervious loose skin, or sheath. It is more resistant to dryness and cold than the egg or the younger larva. However, many of the larvae that become infective die within a few months even in a normal summer, and if the weather is very dry most of them die sooner. Where freezing temperatures occur, a pasture that is given a season's

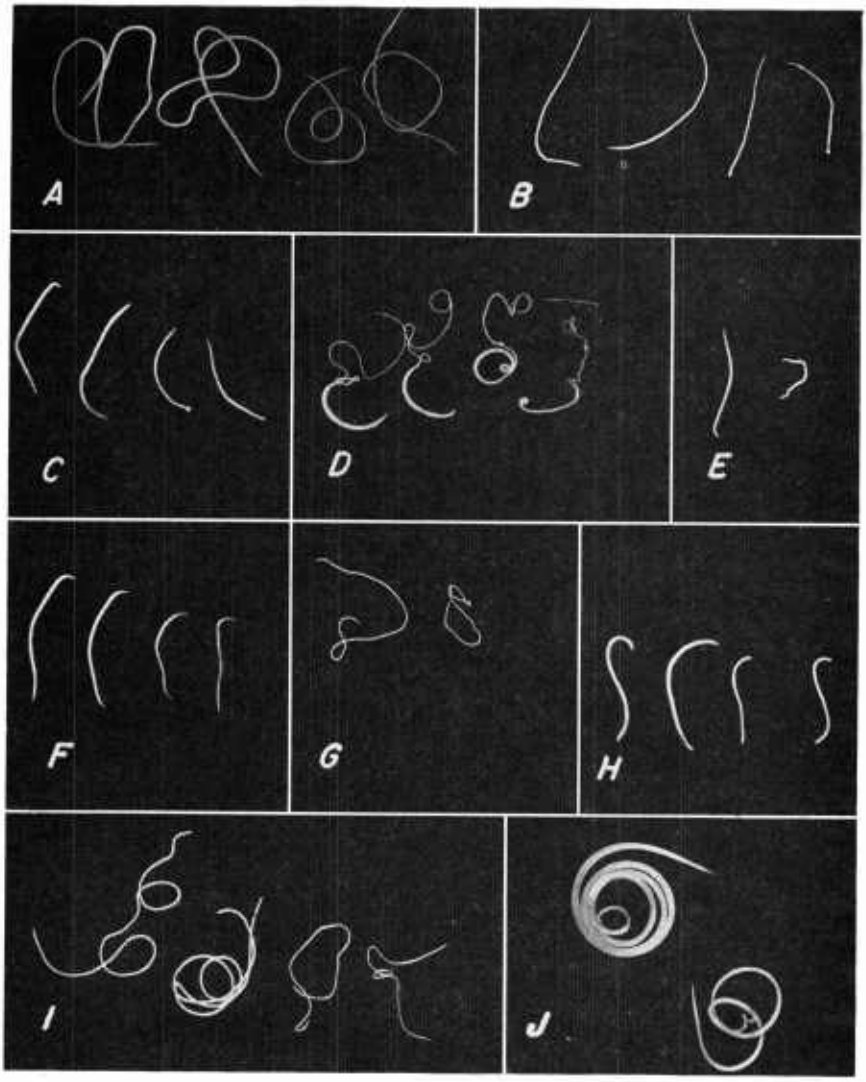


FIGURE 11.—Some of the larger roundworms of sheep (natural size, male or males at the right): *A*, The thread lungworm; *B*, the large stomach worm; *C*, the hookworm; *D*, one of the whipworms, *Trichuris ovis*; *E*, the eyeworm; *F*, the common nodular worm; *G*, the red lungworm; *H*, the large-mouthed bowel worm; *I*, the gullet worm; *J*, the arterial worm.

rest is likely to be free of infective larvae, or nearly so, the following spring.

When the weather is warm and pasture grass is wet with rain or dew, the infective larvae crawl up grass blades and are swallowed by grazing sheep. Within 2 to 3 weeks they develop into mature worms in the stomach and the females begin to produce eggs. However, they usually do not produce large numbers of eggs until somewhat later.

**Distribution.**—This worm is found in almost every place where sheep, cattle, or wild animals such as deer exist, but much more com-

monly in the Eastern, Southern, and Midwestern States than in the Rocky Mountain and Pacific States. It is most prevalent where the summers are warm and wet, least prevalent where they are hot and dry. It is a pest on irrigated pastures.

**Damage and symptoms.**—The large stomach worm kills or injures a great many lambs and sheep, probably more than any other internal parasite. The most characteristic feature of the disease it causes, haemonchosis, is anemia. As the worms develop they damage the stomach lining and cause it to bleed. The adults are bloodsuckers and frequently move from place to place biting the lining and leaving small wounds, from which blood seeps. The amount of blood lost each day by an infested sheep depends largely upon the number of worms it harbors, both young and adult. The number of red cells in its blood may gradually decline until not enough remain to support life.

Fat lambs and even adult sheep that have rapidly picked up heavy infestations may suddenly die. The internal bleeding may be so profuse that it causes their death before they have had time to lose condition. Too often the cause is not discovered until one of the flock dies and large numbers of this worm are found in its stomach. Treatment started then may be too late to save many of the remaining animals of the flock. A good herdsman is on the alert for early signs of acute haemonchosis. He periodically examines his animals for paleness of the eye, the mouth membranes, and the skin, which is a symptom of anemia. An acutely anemic animal appears weak, does not eat normally, lies down a great deal, and staggers when forced to get up. Haemonchosis should be suspected as soon as a diagnosis of anemia is confirmed, and treatment for it should start immediately.

The first symptoms observed in sheep suffering from chronic haemonchosis are unthriftiness, dullness, and listlessness. There may be edema, manifested by swelling of the pendent parts of the body. Such swelling most often affects the part under the jaw, in which case it is called bottle jaw (fig. 12). If not treated, the animals gradually become very thin and may die. Even if they show no marked signs of illness so long as they remain quiet, they may die when driven for a distance.

If the fourth stomach of a sheep infested with the large stomach worm is opened, the worms can usually be seen as wriggling pinkish objects. When the contents of the stomach are poured out, many of the worms usually remain attached to the stomach lining. If they are covered by the stomach contents, a little careful washing will reveal them. Close investigation of the stomach lining may reveal reddish spots, which are punctures, the size of a pinhole, caused by the worm's bites—usually, several for each worm. The carcass of a sheep that has been severely infested with this stomach worm for a long time is likely to be very thin; the meat is pale, and the blood is thin and watery. The heart may be enlarged and flabby, and the fat on the viscera may resemble gelatine.

FIGURE 12.—Bottle jaw, caused by the large stomach worm.

**Treatment and control.**—The large stomach worm can be controlled by following sound management practices. Give special attention to young stock. Graze healthy, clean animals on clean pastures; eliminate or fence off low, swampy areas; rest pastures occasionally; avoid overstocking; segregate sick animals promptly; introduce only clean stock; provide good feed at all times, supplementing pasturage when necessary; and use drugs as outlined here. Rest periods for sheep pastures are an effective means of controlling the large stomach worm because the worm cannot overwinter on idle pastures except in numbers so small as to be insignificant.

Several effective treatments are available for removing large stomach worms. In the order of overall usefulness, these are (1) phenothiazine, (2) copper sulfate and nicotine sulfate, (3) copper sulfate, (4) carbon tetrachloride, (5) tetrachlorethylene, and (6) hexachloroethane. Most of them act effectively not only against the large stomach worm but also against certain other parasites. This is fortunate, since sheep infested with the large stomach worm are likely to be infested with other parasites also. The other parasites against which these treatments are effective will be named in the discussions of the different treatments.

None of these treatments requires fasting or purging of the animals.

*Phenothiazine.*—Phenothiazine, first tested against sheep parasites in 1939, has now been widely and successfully used in all the important sheep-raising areas of the world, on countless thousands of animals. No other drug compares with it for use in sheep as a general worm remedy.

Phenothiazine acts effectively not only against the large stomach worm but also against several other highly injurious species of roundworms that are parasites of the stomach and intestines. These include the medium stomach worms, stomach and intestinal hairworms, the hookworm, the large-mouthed bowel worm, and the nodular worm. The drug also has limited value for control of the cooperias, thread-necked worms, and the whipworms. It has no effect on capillarids, tapeworms, liver flukes, or protozoa.

The drug is commonly given in doses of 25 grams (about 1 ounce) each to adult sheep and 15 grams each to lambs under 60 pounds. In different areas, the doses that are commonly used vary approximately from 20 to 40 grams for adult animals and from 10 to 20 grams for lambs.

Two treatments of the breeding flock, one in the fall or early winter at least a month before lambing and the other in the spring after lambing but before the grazing season begins, are often enough, especially if phenothiazine is given by the free-choice method during the grazing season.

The stomach hairworm and the intestinal hairworms, which are sometimes very numerous and exceedingly injurious, are not controlled satisfactorily with any dosages of phenothiazine smaller than 35 to 40 grams (about 1⅓ ounces) for adult animals and 20 grams (about two-thirds ounce) for lambs. The larger dosage should therefore be used when symptoms or other findings indicate infestation with hairworms.

Individual doses may be given as drenches, in capsules, or in the feed. Several animals may be treated at a time by mixing the right amount of drug into the feed. Commercially prepared boluses (large pills) and tablets are satisfactory if they disintegrate rapidly in the sheep's digestive tract.

Extremely few instances are known of injury to sheep from medication with phenothiazine, but the drug, being a dye, stains the fleece. Ordinarily this is not an

important factor, but staining can be reduced by keeping the sheep on thick bedding for 3 or 4 days, or on ground where the urine will be absorbed quickly.

Many sheepmen give phenothiazine to their flocks by the free-choice method, in loose salt or mixed minerals, with satisfactory results. Provide a dry mixture containing 1 part, by weight, of phenothiazine and from 9 to 14 parts, by weight, of loose salt or other mineral supplement. Shelter the medicated mixture from the weather and replenish it regularly, keeping ample amounts before the sheep at all times. Each animal should get about one-half gram of phenothiazine a day. This amount largely prevents development of infective parasite larvae in the manure. The drug may be given in this way continuously or only during the grazing season. When the latter method is used, it should be combined with full doses of the drug to breeder stock during the winter, as outlined above.

The free-choice method of giving phenothiazine is not foolproof and cannot be used under all circumstances. Also, it does not take the place of good management. The medicated mixture must contain the only salt or other mineral supplement that is available to the flock. For this reason, the method cannot be recommended for use in areas where natural salt is present. The containers must be large and numerous enough to permit all animals in a flock to partake at will. Put the mixture near the common water supply, and preferably also near the customary feeding place. Add a small amount of grain or chopped alfalfa to the mixture at least until the sheep become familiar with it. The program must be started on the first day of the season, when sheep are first turned into spring pastures. Do not wait until the pastures have become contaminated or sheep have been found to be infested. The mixture must be kept loose and uncaked. Finally, watch for any animals that require special medication, particularly if some do not take the mixture.

Use of medicated salt blocks may not be a reliable or economical practice; the animals usually do not get enough of the drug for control of the parasites. However, properly formulated, soft-textured blocks, adequately sheltered, may yet prove useful.

*Copper sulfate and nicotine sulfate (Cu-Nic).*—Copper sulfate and nicotine sulfate in combination (Cu-Nic) form a satisfactory drench against large stomach worms comparable in effectiveness to copper sulfate alone and also act rather effectively against hairworms and common intestinal tapeworms. This treatment, like phenothiazine, is one of the few that give fairly good results against medium stomach worms and thread-necked worms.

To prepare a 1-percent Cu-Nic drenching solution, add 1 ounce of commercial nicotine sulfate solution (Black Leaf 40) to 1 gallon of a 1-percent solution of copper sulfate. Copper sulfate solutions must not be prepared in metallic containers unless the containers are coated with enamel or porcelain, and only clear blue crystals of the compound should be used. To make a 1-percent solution, dissolve one-quarter pound of copper sulfate in 1 pint of boiling water, then add cold water to make a total of 3 gallons. To make a 2-percent Cu-Nic drench, add 2 ounces of commercial nicotine sulfate solution to each gallon of a 2-percent solution of copper sulfate, which is prepared by dissolving one-half pound of copper sulfate in 1 quart of boiling water and then adding water to make 3 gallons.

The 1-percent drench is ordinarily given in doses of 2 to 3 ounces for adult sheep, 1½ to 2 ounces for yearlings, and ½ to 1 ounce for

lambs. The 2-percent drench may be given at the rate of ½ to 2 ounces per animal over 3 months.

This treatment is not well tolerated by animals in poor condition. In apparently healthy animals it often causes temporary setbacks and sometimes even causes death. Its range of action, however, offsets these disadvantages and makes it generally more useful than copper sulfate alone.

*Copper sulfate.*—Copper sulfate, in the form of the familiar bluestone drench, is the oldest and cheapest of treatments for destroying the large stomach worm. It effectively removes mature stomach worms from about 8 or 9 out of 10 infested sheep; it does not affect the immature worms. It may act to some extent against the broad tapeworms. In this country, the 1-percent solution has been widely used. In some countries, 2- and 4-percent solutions are preferred. With regard to safety, effectiveness, and convenience, a 2-percent solution may be the best for routine treatment against stomach worms in average-sized sheep. Approximate dosages recommended for different solutions are given in table 1.

Solutions of 1, 2, and 4 percent may be prepared by using ¼, ½, and 1 pound, respectively, of copper sulfate for each 3 gallons of drench. Dissolve the bluestone crystals in a small amount of boiling water and add cold water to the amount needed.

In general copper sulfate, or any chemical given together with it or immediately after it, goes directly to the fourth stomach, where it acts on parasites.

Animals in which copper sulfate does not act should be treated with phenothiazine or carbon tetrachloride.

*Carbon tetrachloride.*—Carbon tetrachloride gives good results when used against the large stomach worm in old sheep and in other sheep that do not respond to copper sulfate. Its action is not dependent on direct passage to the fourth stomach; hence, there is no need for a preliminary drench with dilute copper sulfate solution. It may have some effect against the hookworm and one of the cooperias, *Cooperia curticei*. Against the large stomach worm, the individual dose for an average-sized animal is 2 cubic centimeters for an adult sheep and half that amount for a lamb. The chemical is given either in capsules or as a drench. The drench is usually prepared by dissolving the carbon tetrachloride in from 2 to 5 parts of mineral oil.

For unknown reasons, sheep occasionally die as a result of being treated with carbon tetrachloride. However, the danger of carbon tetrachloride poisoning is not greater when the drug is given in doses of 2 cubic centimeters than when doses half as large are given, as recom-

TABLE 1.—*Dosages of copper sulfate solution for use in treating sheep infested with the large stomach worm*

| Strength of solution | Adult sheep [1] | | Yearlings and large lambs | | Lambs under 6 months | |
|---|---|---|---|---|---|---|
| | *Fluid ounces* | *Cubic centimeters* | *Fluid ounces* | *Cubic centimeters* | *Fluid ounces* | *Cubic centimeters* |
| 1 percent | 2½ to 4 | 75 to 120 | 1½ to 2½ | 45 to 75 | ½ to 1 | 15 to 30 |
| 2 percent | 2 | 60 | 1 to 1½ | 30 to 45 | ½ to ¾ | 15 to 25 |
| 4 percent | 1 to 2 | 30 to 60 | ½ to ¾ | 15 to 25 | | |

[1] For especially large mature sheep, the maximum dosages shown should be increased by about 20 percent.

mended later for animals infested with liver flukes.

*Tetrachlorethylene.*—In doses of 5 cubic centimeters for adult sheep and half that much for lambs, tetrachlorethylene is comparatively safe and as a rule acts effectively not only against the large stomach worm but also against hairworms and the hookworm. It may be given in mixture with 1 to 2 parts of mineral oil as a drench.

Accidents in dosing are more frequent with tetrachlorethylene than with some of the other drugs discussed here, on account of inhalation. Instances of poisoning are common but rarely serious. To serve as a worm remedy, the drug must go directly to the fourth stomach. It must therefore be preceded by a small copper sulfate drench, such as 10 cubic centimeters (1/3 ounce) of a 1-percent solution. Tetrachlorethylene shows little or no efficacy in some cases, even if preceded by a dose of copper sulfate.

*Hexachloroethane.*—Hexachloroethane acts effectively against stomach worms. Its safety as a treatment is about the same as that of carbon tetrachloride. A dose of 60 cubic centimeters (2 ounces) of a hexachloroethane - bentonite - water suspension, containing 30 grams of hexachloroethane, is given to an adult sheep, and half that dose to a lamb.

### MEDIUM STOMACH WORMS

**Location.**—The medium, or brown, stomach worms live in the fourth stomach, generally in the end nearer the small intestine. They are found in the small intestine occasionally.

**Appearance.**—These worms (fig. 13, *B*) are brownish, hairlike, and about one-half inch long.

**Life history.**—Direct. The free-living development resembles that of the large stomach worm. Infective larvae of the commonest species, when swallowed by sheep, penetrate the lining of the stomach and develop in it for about a week. The larvae then normally emerge into the cavity of the stomach, where they become mature adults about 15 to 17 days after infection. Sometimes they do not emerge until they have become mature or nearly so. In the stomach cavity, both immature and mature worms live in very close contact with the lining.

**Distribution.**—*Ostertagia circumcincta*, the commonest species, and *O. trifurcata*, also common, are widely distributed. Three other species, which have a restricted distribution, are mentioned later. In the West, the medium stomach worms affect more sheep than the large stomach worm.

**Damage and symptoms.**—As a result of penetration by the larvae parts of the stomach wall become inflamed, dotted with small white elevations, and marked with minute hemorrhages. As the worms grow, the elevated areas increase in size and become nodules (fig. 14). The damage appears to interfere with normal digestion. When the worms emerge from them, the nodules recede and disappear.

Sheep heavily infested primarily with medium stomach worms progressively lose weight and condition, have a stunted appearance, scour intermittently, show excessive thirst, and become anemic. Death rather often results. None of these symptoms are peculiar to infestation with these worms, and sheep having medium stomach worms usually are infested also with the large stomach worm and one or more of the common roundworms of the intestine.

**Treatment and control.**—Phenothiazine and the Cu-Nic drench, used as recommended for removal of the large stomach worm (pp. 16, 17), are the best available treatments against medium stomach worms. These worms are harder to destroy than the large stomach worm, and neither treatment has always proved

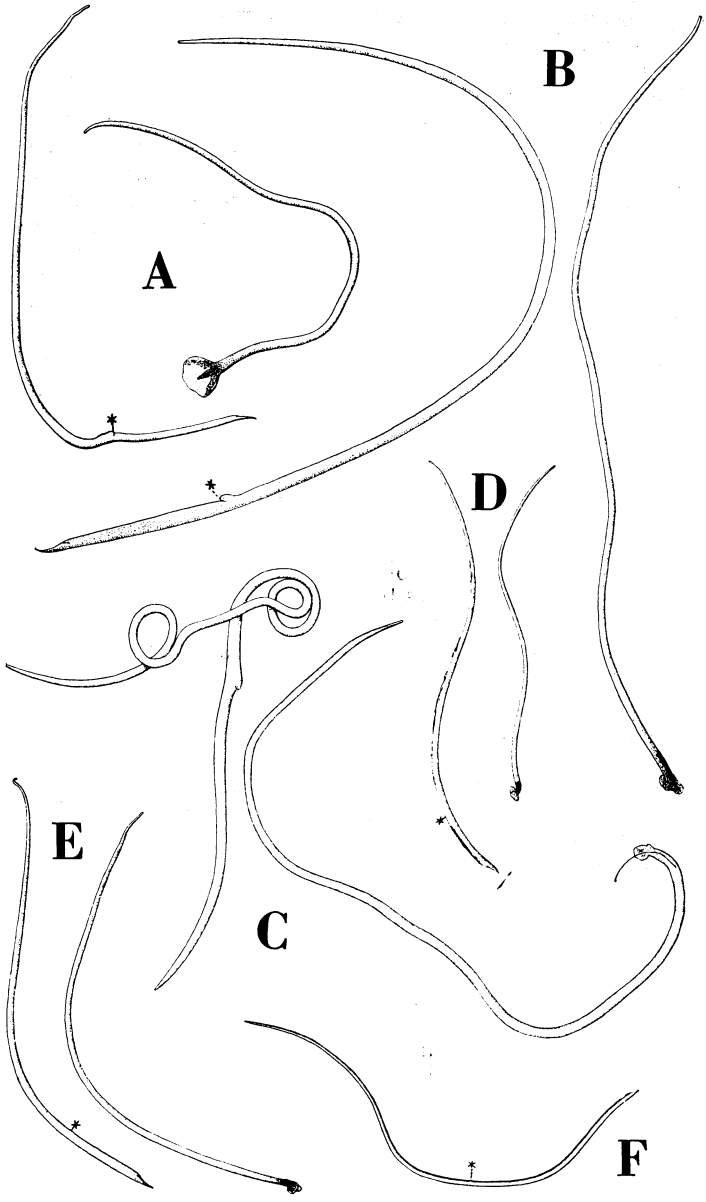


FIGURE 13.—Some common smaller roundworm parasites of sheep (magnified 13 times; male of each pair at the right): *A*, One of the cooperias, *Cooperia oncophora; B*, one of the medium stomach worms, *Ostertagia circumcincta; C*, one of the thread-necked worms, *Nematodirus spathiger; D*, the stomach hairworm; *E*, one of the intestinal hairworms, *Trichostrongylus colubriformis; F*, the threadworm (female; all the parasites are of this sex).

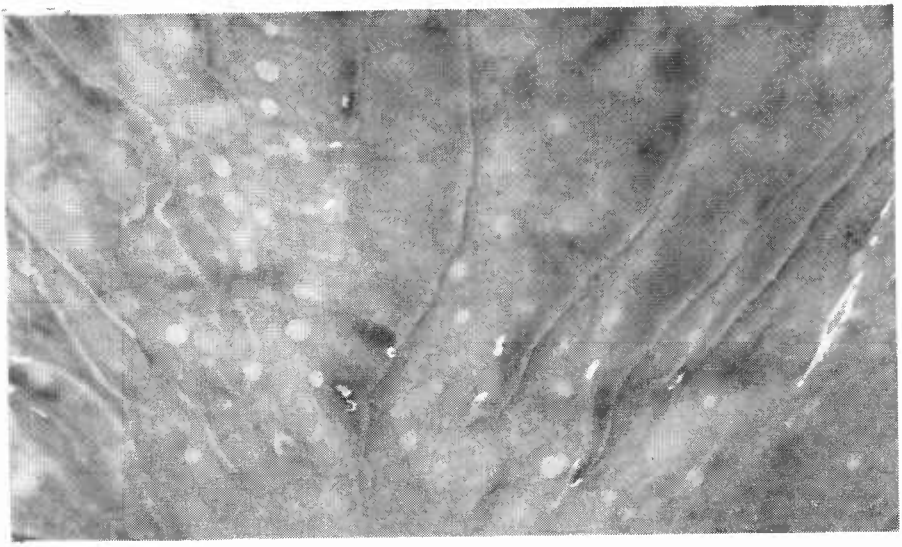

FIGURE 14.—Part of a sheep stomach showing nodules on wall caused by medium stomach worm larvae.

satisfactory against them under outbreak conditions. Repeat treatment at intervals of 2 to 3 weeks. Use the control methods advised for the large stomach worm.

## STOMACH HAIRWORM

**Location.**—The stomach hairworm lives primarily in the fourth stomach. It can live in the upper small intestine, also.

**Appearance.**—The worms (fig. 13, *D*) are extremely small and are not likely to be seen in the opened stomach even if present in large numbers. They are only about one-fourth inch long and are as fine as the finest hair. But if the lining of an infested stomach is scraped and the scrapings are mixed with clear water and examined in a glass dish on a dark background, the tiny, wriggling, whitish, translucent worms can be seen.

**Life history.**—Direct. The free-living development is similar to that of the large stomach worm. Infective larvae swallowed by a sheep migrate into the pits in the lining of the stomach. After a period of development there, the worms move out into the cavity of the stomach and there become sexually mature adults about 3 weeks after infection. After an egg of this species passes onto the pasture and an embryo develops in it, it is much more resistant to the effects of drying than eggs of most other roundworms of sheep are at any stage. The eggs can survive a long drought and give rise to infective larvae when the drought is ended by rain, whereas even short spells of dry weather may kill the eggs of some of the other common roundworms.

**Distribution.**—This stomach worm is widely distributed and rather commonly infests not only sheep and goats but also horses and cattle.

**Damage and symptoms.**—Stomach hairworm infestations cause the stomach lining to become inflamed and in some cases to become ulcerated. Heavy infestation may cause lambs to die, particularly if their feed is poor. Death may be preceded by a severe watery diarrhea that causes the animal's body to dry out and become emaciated. Loss of appetite, roughness of the fleece, and stunting also may be noted (fig. 15). No symptom can

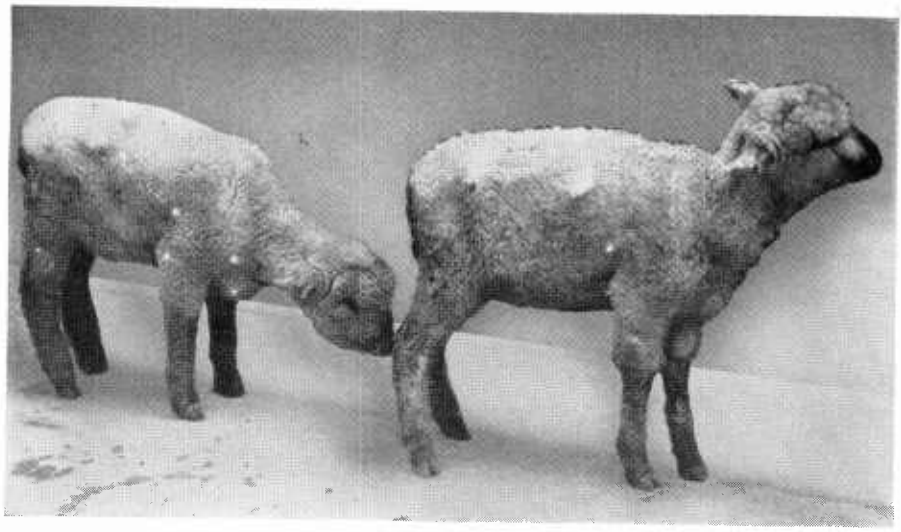

FIGURE 15.—Lambs in an advanced stage of trichostrongylosis due to the stomach hairworm.

be named that is peculiar to infestation with this worm. Disease caused by the stomach hairworm (or by intestinal hairworms, discussed in the next section) is called trichostrongylosis.

**Treatment and control.**—The same treatment is recommended as for medium stomach worms. Tetrachlorethylene, used as against the large stomach worm (p. 19), may have value. Control measures are the same ones recommended for the other stomach worms.

## INTESTINAL HAIRWORMS

**Location.**—Three species of hairworms, *Trichostrongylus colubriformis*, *T. vitrinus*, and *T. capricola*, live in the duodenum (the upper part of the small intestine) and occasionally in the fourth stomach.

**Appearance.**—These worms (fig. 13, *E*) are about one-third inch long and about as fine as a fine hair. To find them, scrapings from the lining of the intestine should be mixed with clear water and examined in a glass dish on a dark background.

**Life history.**—Direct. The free-living development resembles that of the large stomach worm. Infective larvae of the commonest species when swallowed by a sheep penetrate into the wall of the small intestine. The young worms develop there for a few days, then return to the intestinal cavity and develop into adults, which reach sexual maturity about 17 days after infection. The eggs are highly resistant to drying after the embryo develops.

**Distribution.**—All three species are widely distributed. *T. colubriformis* is the most common. *T. capricola* is less frequent in sheep than in goats.

**Damage and symptoms.**—On post mortem examination of animals affected with trichostrongylosis caused by intestinal hairworms, no noticeable injuries appear in the intestines. The first 10 feet of the small intestine may contain an abnormally large amount of mucus, and its wall may be congested. Usually, the muscles have wasted away, fat on the viscera is almost completely lacking, and the visceral fat that remains is swollen and gelatinous.

The outstanding symptom is a more or less persistent diarrhea.

Other symptoms include loss of appetite, progressive weakening, emaciation, and stunting. Although the name "black scours" is sometimes applied to this disease, the manure is not dark colored in uncomplicated cases. The conditions commonly called bottle jaw and "potbelly" are absent. The course of the disease is strongly influenced by the quantity and quality of available feed. Particularly in dry summers, when the grass is short, death is likely to result.

Typically, trichostrongylosis caused by intestinal hairworms is a slow, long-drawn-out disease that chiefly affects animals 3 to 18 months old. Often it appears soon after weaning and continues through the summer and the next winter. The droppings are soft and pasty at first, but may rapidly become fluid and watery as the disease progresses. The scouring may last for weeks or even months. Occasionally the bones of heavily infested animals weaken, with the result that the animals become lame and their leg bones break easily. Sometimes the disease kills yearlings, as well as lambs.

Occasionally, animals die of trichostrongylosis within 2 to 4 weeks after they have suddenly taken in very large numbers of infective intestinal hairworm larvae and hence after a rather short period of very severe scouring.

When sheep become heavily infested with the large stomach worm and stomach and intestinal hairworms, as they commonly do, they not only scour but also become anemic.

**Treatment and control.**—Phenothiazine in doses of 35 to 40 grams for adult sheep and 20 grams for lambs under 60 pounds is the most dependable treatment against intestinal hairworms. Secondary choices are the Cu-Nic drench and tetrachloroethylene, given as described for the large stomach worm. For checking outbreaks of trichostrongylosis, repeated treatments at intervals of 2 to 3 weeks may be necessary. Control methods are the same as those recommended for the large stomach worm. Observations in Australia have shown that good forage tends to prevent severe outbreaks of trichostrongylosis.

## COOPERIAS

**Location.**—The cooperias inhabit the small intestine.

**Appearance.**—The cooperias (fig. 13, *A*) are hairlike worms about one-quarter to one-half inch long, brownish red when alive. Their presence is not readily detected unless scrapings from the lining of the small intestine are mixed with clear water and examined in a glass dish on a dark background.

**Life history.**—Direct. The free-living development resembles that of the large stomach worm (p. 13). Infective larvae of the commonest species when swallowed by a sheep penetrate deeply into the spaces between the small intestinal villi (minute, fingerlike projecting parts of the intestinal lining), but they do not invade the intestinal lining. The young worms soon move out into the cavity of the intestine, and there they develop into sexually mature adults in about 2 weeks.

**Distribution.**—*Cooperia curticei* is widely distributed, and infestations with it are very common. *C. oncophora* is distributed principally in the North. Wherever it occurs, infestations with it are common. *C. punctata* and *C. pectinata* infest sheep infrequently and are most likely to occur in those that are grazed with cattle.

**Damage and symptoms.**—The intestinal lining of a sheep repeatedly infected with larvae of *C. curticei* becomes inflamed. Nodules form in it, each of them enclosing a young worm. Although there is evidence that anemia and symptoms similar to those characteristic of trichostrongylosis are associated

with natural cooperia infestations, experimental infestations with the commonest species have had only one important effect on sheep—reduction of ability to gain weight on good feed. The possibility that any of the other species, or any mixture of two or more of the species, may have more severe effects has not been tested.

**Treatment and control.**—No satisfactory treatment is known. Carbon tetrachloride, used as for treatment and control of the large stomach worm (p. 18), may be of some value against *C. curticei*. Doses as large as 3 cubic centimeters have been used against this cooperia. Phenothiazine, given by the free-choice method as recommended for stomach worms (p. 17), may have some preventive value. Chief reliance must be placed on general preventive practices.

## HOOKWORM

**Location.**—The hookworm lives mainly in the middle part of the small intestine.

**Appearance.**—Usually the females are about 1 inch and the males about ¾ inch long (fig. 11, *C*). The body is a little smaller in diameter than an ordinary pin and is whitish to pinkish. The head end of the body is slightly hooked.

**Life history.**—Direct. The free-living development resembles that of the large stomach worm. Infection of sheep takes place both by way of the mouth and through the skin. Infective larvae that get onto the skin penetrate it and pass inward to the lungs. After a brief period of development there, the young worm migrates to the small intestine and attaches itself to the intestinal lining by means of its mouth. Here it develops into an adult, which slowly reaches sexual maturity. Eggs appear in the manure about 8 weeks after infection. When infective larvae are swallowed, apparently only a small percentage of them penetrate the lining of the digestive tract, pass to the lungs, return to the digestive tract, and give rise to adult worms in the small intestine.

**Distribution.**—The hookworm is widely distributed. Infestations are fairly common but are seldom heavy.

**Damage and symptoms.**—Loss of blood from the lining of the intestine begins when the developing worms reach that organ, about 3 weeks after infection. The adult worms, and probably the younger ones, bite into the lining of the intestine to obtain blood from the capillaries. They move frequently from place to place on the lining to feed, and each puncture bleeds for a time after the worm moves to a fresh spot. A moderately or heavily infested sheep loses so much blood that it becomes anemic. The thinned, anemic blood may seep out of the blood vessels and into the tissues, causing watery swellings of pendent parts of the body and a dropsical condition generally through the body. The impoverishment of the blood impairs the animal's nutrition—a very serious matter, particularly with young animals. Symptoms of hookworm disease include paleness of the mucous lining of the eyelids and mouth, pale skin, dry wool, watery swellings under the jaw and along the abdomen, and general unthriftiness.

From symptoms alone, it is practically impossible to differentiate hookworm disease from the chronic form of haemonchosis, the disease caused by the large stomach worm. However, a post mortem examination of the fourth stomach and the small intestine will show which of the worms is present, or whether both are present. Lesions caused by the hookworm appear as red spots in the small intestine; similar spots in the fourth stomach are caused by the large stomach worm.

Hookworm infestations of sheep severe enough to cause death by themselves apparently are rare in this country. However, combined infestations with the large stomach worm and the hookworm are fairly common. In some of these the hookworms make the blood loss materially greater than it would be if only the stomach worms were present, and probably the drain they add is sometimes the cause of death. Lambs on a good diet withstand hookworm infection and infestation better than those on a poor diet, particularly one deficient in iron and protein.

**Treatment and control.**—Standard doses of phenothiazine, 25 grams for adult animals and 15 grams for lambs, are the most reliable medication against hookworms. Other remedies sometimes used include tetrachlorethylene, carbon tetrachloride (given as described on p. 18), and Cu-Nic drench (given as described on p. 17). The only control measures known are those that have been described for the large stomach worm. Good feed helps to minimize the injurious effects. Because this worm enters the sheep through the skin as well as through the mouth, boggy land and loose, wet, sandy soil provide conditions that favor infection.

### THREAD-NECKED WORMS

**Location.**—The thread-necked worms inhabit mainly the upper half of the small intestine.

**Appearance.**—The worms (fig. 13, *C*) are pinkish and rather slender. The length of the females is about 1 inch or a little less, that of the males about three-quarter inch. The body of the female tapers more abruptly toward the head than toward the tail.

**Life history.**—Direct. The infective larvae of the thread-necked worms develop within eggs that have passed out in the manure. Under favorable conditions they complete their development in from 10 days to 2 weeks. When they hatch, usually some days or weeks later, they crawl up onto grass blades and other forage. Both the eggs and the infective larvae are especially resistant to cold and drying. When swallowed by sheep, these larvae develop into sexually mature adult worms in the small intestine. For the commonest species, this process usually takes about 3 weeks.

**Distribution.**—Two of the thread-necked worms, *Nematodirus spathiger* (the commonest species) and *N. filicollis*, are widely distributed; a third species, *N. abnormalis*, occurs mostly in the North Central States. Thread-necked worm infestations are very common.

**Damage and symptoms.**—The young worms damage the intestinal lining. In experiments, thread-necked worm infestations in lambs have produced effects similar to those caused by intestinal hairworms—unthriftiness, retardation in growth, and intermittent diarrhea. However, the damage was less severe and did not cause death.

Under field conditions, thread-necked worms apparently are the primary cause of some outbreaks of scouring that result in unthriftiness, weakness, emaciation, and rather high death losses. Apparently they do great damage when they are the chief element in heavy mixed gastrointestinal roundworm infestations. In experiments, lambs given both thread-necked worms and large stomach worms, but not enough of the latter to cause serious anemia by themselves, scoured persistently and became very anemic; one died. Lambs given both intestinal hairworms and thread-necked worms were much more severely affected than those given either of these kinds of worms alone.

**Treatment and control.**—Phenothiazine, carbon tetrachloride, tetrachlorethylene, hexachloroethane, and the Cu-Nic drench have been

used as treatments for removal of thread-necked worms, but these drugs have not proved reliable in single doses. Large doses of phenothiazine or repeated doses of the other drugs have sometimes been credited with giving better results than single doses of the usual size. Phenothiazine given by the free-choice method controls the infestation to a considerable extent. Overwinter resting of pastures does not materially reduce the season-to-season carryover, for the larvae can withstand cold weather. Hot-season resting of pastures gives better results. So far as possible, the sheepman should prevent buildup of other parasites, such as stomach worms and intestinal hairworms, in animals infested with thread-necked worms.

### THREADWORM

**Location.**—The threadworm lives in the small intestine, chiefly in the upper part.

**Appearance.**—The parasitic worms (fig. 13, *F*), all females, are about as fine as a fine hair and only about ⅛ to ¼ inch long; they can be found by mixing scrapings from the lining of the small intestine with clear water and examining them in a glass dish on a dark background.

**Life history.**—Direct. The eggs, each already containing an embryo when it passes out in the manure, hatch in a few hours under favorable conditions. Usually two different types of development then occur. Many of the larvae develop directly to the infective stage in about 2 days. Meanwhile, the others develop into very small free-living male and female worms. Then, the eggs produced by the free-living females give rise to infective larvae in about 2 days.

The infective larvae, unlike those of other roundworms infesting sheep, are not ensheathed. They are extremely susceptible to drying and cold. Like hookworm larvae, they can infect a sheep by way of either the skin or the mouth. After penetrating the skin or, if swallowed, the wall of the digestive tract, the larvae migrate in the body, reach the small intestine, and there transform into mature females. This parasite develops very rapidly; eggs appear in the manure 7 days after infection.

**Distribution.**—The threadworm is widely distributed. In many parts of the country scarcely a lamb escapes infestation. The infestations are usually rather light, however.

**Damage and symptoms.**—The passage of infective larvae through the skin sometimes causes an inflammation of the skin. If this happens between the toes, it increases the chance that the sheep may get foot rot. The bacteria that cause foot rot pass more readily through diseased than through healthy skin.

Intestinal threadworm infestations cause no characteristic symptoms. Typical infestations seem to have little if any effect on the sheeps' health. However, damage to the lungs and intestinal lining, diarrhea, frequent urination, emaciation, mild anemia, and death can result from heavy infestations, especially in lambs.

**Treatment and control.**—No treatment or specific measures of control can be recommended.

### COMMON NODULAR WORM

**Location.**—The adults of the nodular worm live in the cecum (the pouch in which the large intestine begins) and the colon. Normally the larvae of this worm temporarily invade the walls of the large and small intestines. Occasionally they reach other viscera and tissues. Very conspicuous nodules form about the larvae if they remain in the tissues for a long time. These nodules are found most frequently in the wall of the large intestine, but they may be present throughout the length of the intestinal tract.

**Appearance.**—The female worms grow to a length of about five-eighths inch; the males are a little shorter (fig. 11, *F*). Both are whitish and rather stout. The forward part of the body is hooked.

**Life history.**—Direct. The free-living development resembles that of the large stomach worm. Infective larvae swallowed by a sheep enter the walls of the small and large intestines, where small cysts form about them. In a sheep that has been infected for the first time, many of the larvae soon vacate these cysts, pass into the intestinal cavity, and develop into sexually mature worms in the colon and cecum about 7 to 8 weeks after infection. Others usually remain in the gut wall for several weeks. In a sheep that has been infected repeatedly, some larvae may remain in the gut wall for several months. The nodules from which the parasite has received its name form about any larvae that remain in the cysts for more than a week or so, move more deeply into the gut wall, or migrate elsewhere in the viscera. Many of the nodules in the gut wall open into the intestinal cavity, and the larvae in them may in the course of time escape into it. The young worms in nodules that have no opening into the intestinal cavity, and those located in such organs as the lungs or liver, die in the nodules.

**Distribution.**—This parasite is distributed over the Atlantic, North Central, and South Central States. Infestations are common in these regions, especially in the New England and Middle Atlantic States.

**Damage and symptoms.**—In some places the damage done by this worm has caused sheep raising to be abandoned. When the larvae remain in the gut wall for a long time, or move inward, they set up a severe local tissue reaction. This results in formation of nodules surrounding them (fig. 16), which may become as large as a good-sized pea. At first these contain a mass of

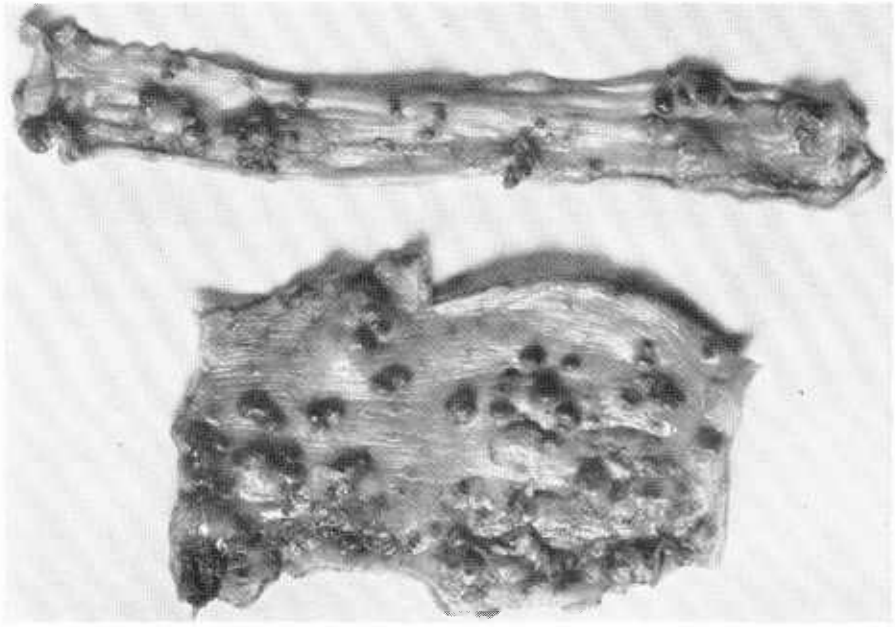

FIGURE 16.—Outer surfaces of parts of small (above) and large intestines of a sheep showing nodules caused by larvae of the common nodular worm.

greenish creamy material, which soon becomes cheesy in consistency and later becomes hard and stonelike. Wandering larvae leave tracks filled with similar material. When the nodules are large and numerous, they interfere with the functioning of large areas of the intestines. They may also obstruct an intestine to an extent sufficient to block it or cause telescoping of parts of it.

The larvae and adult worms in the cavity of the intestine irritate and inflame the intestinal lining. They, and also the bacteria that get into the nodules, may form substances that have a poisonous effect on the host.

Nodular-worm disease seldom causes death, but it retards growth, lowers the quality of mutton, lamb, and wool, and impairs the value of breeding stock. Nodular intestines, or "knotty guts," are unfit for use in producing sausage casings or surgical sutures. Some infested sheep harbor few adult worms but have many nodules, some have few nodules but harbor many adult worms. The symptoms of infestation include poor appetite, failure to gain properly or slight to pronounced loss of weight, diarrhea, wool damage, and mild anemia. Because these symptoms are similar to those caused by other worm infestations, the diagnosis must be made by post mortem examination or by laboratory identification of worm eggs in the manure.

**Treatment and control.**—Treatment is essential to the control of nodular-worm disease. Nothing cures it. Young worms in nodules in the intestines are beyond the reach of drugs, and no treatment removes the nodules. Phenothiazine, used as recommended for removal of the large stomach worm (p. 16), effectively removes adult nodular worms from the large intestine. Removal of the adult nodular worms immediately benefits diseased sheep to some extent and also helps to prevent more serious disease in these and other animals by reducing the risks of infection. Other important control measures are pasture hygiene and general sanitation.

In the Northern States, sheepmen can largely protect their flocks against infestation with the common nodular worm by taking advantage of the fact that the long, hard winters almost eliminate pasture contamination. At the end of a moderately severe winter, none of the infective larvae survive on an unused pasture. Therefore, little infection with this parasite is carried over to another season except in the breeding stock. The ewe is the most dangerous source of infection to the young lamb. Ewes should be treated at least once during the winter, preferably twice with an interval of at least a month between treatments, in order to remove as many of the parasites as possible.

### LESSER NODULAR WORM

**Location.**—The lesser nodular worm, closely related to the common nodular worm, lives chiefly in the cecum.

**Appearance.**—The adults tend to be a little larger than adults of the common nodular worm, but cannot readily be distinguished from them without a microscope.

**Life history.**—Direct. The free-living development resembles that of the large stomach worm. Infective larvae swallowed by a sheep invade the wall of the small intestine. The young worms return to the intestinal cavity in a few days, pass to the cecum, and there develop into adult worms. Eggs appear in the host's manure about 28 days after infection.

**Distribution.**—The range of this worm extends somewhat farther west than that of the common nodular worm; otherwise, the two species have about the same range. The common nodular worm is the more numerous in most infestations.

**Damage and symptoms.**—While the larvae are in the wall of the small intestine, small cysts form around them; these heal and disappear soon after the larvae vacate them. Large, hard nodules are rarely, if ever, produced by larvae of this worm. Infestations may produce small hemorrhages in the wall of the cecum. They can cause soft or semifluid droppings with excessive amounts of mucus, occasionally flecked with blood, or a mild, temporary diarrhea beginning about 2 weeks after infection and sometimes continuing about 2 weeks. A moderate to heavy infestation decreases the rate at which the host animal gains weight.

**Treatment and control.**—Follow measures recommended for treatment and control of the common nodular worm.

### LARGE-MOUTHED BOWEL WORM

**Location.**—The large-mouthed bowel worm usually inhabits the coiled part of the colon.

**Appearance.**—The maximum length of the worm is nearly 1 inch; the males are a little smaller than the females (fig. 11, *H*). This parasite resembles the nodular worms. However, the head, which is slightly hooked to one side, is larger than the neck. The mouth is cup-shaped.

**Life history.**—Direct. The free-living development resembles that of the large stomach worm. Infective larvae swallowed by a sheep enter the wall of the upper colon. After a few days the young worms return to the cavity of the colon, and there they develop into adult worms. They reach sexual maturity about 7 to 8 weeks after infection.

**Distribution.**—This worm has about the same range as the common nodular worm, but it is the less common of the two in most localities.

**Damage and symptoms.**—The developing larval worms can cause the colon to become congested and its wall to become thickened and studded with small hemorrhages. Also, they may destroy much of the lining of the colon. The adults can cause similar damage and production of bloody mucus, but the congestion they cause is patchy. The adult worm sucks up a small tuft of the lining of the colon and feeds on it. Then, having largely destroyed that tuft, it moves on to another.

The symptoms include diarrhea, usually intermittent, with blood and mucus in the droppings, marked loss of weight, and moderate anemia. They appear about 1 to 2 months after infection and seem to be caused mainly by the immature worms. Infestations may cause death.

**Treatment and control.**—Standard doses of phenothiazine, 25 grams for adult animals and 15 grams for lambs under 60 pounds, are the most reliable means of combating infestations of the large-mouthed bowel worm. The only control measures known are those that have been described for the large stomach worm.

### WHIPWORMS

**Location.**—The cecum is the special habitat of the whipworms.

**Appearance.**—These worms (fig. 11, *D*) are whitish and about 2 to 3¼ inches long. The males tend to be a little larger than the females. The forward ⅔ to ⅘ of the body is exceedingly slender, and only the remaining rear part, which is very much thicker, can readily be seen against the background of the gut wall. The long, slender forward part may be compared to a whiplash and the short, thick part to a whipstock or handle; hence the name "whipworm."

**Life history.**—Direct, so far as known. In each of the eggs carried out with the manure, an infective larva develops in about 2 weeks, if weather conditions are very favorable. The eggs never hatch in the

open. Beyond this point, the life history is not fully known for any of the whipworms of sheep. In all probability, it is as follows: Infective eggs swallowed by a sheep hatch in the digestive tract, and the larvae invade the wall of the small intestine. The young worms later reenter the cavity of the intestine and there develop into adults. These move to the cecum, where they become sexually mature.

**Distribution.**—The common species, *Trichuris ovis*, is widely distributed. Two others, *T. globulosa* and *T. discolor*, occur, but their distribution is unknown. Most sheep harbor whipworms at some time.

**Damage and symptoms.**—The worms usually are found with their slender forward ends rather deeply buried in the lining of the cecum. Inflamed, thickened areas sometimes surround the points at which they are attached. Whipworm infestations have not been shown to have an appreciable effect on the health of sheep or to cause well-defined symptoms. The presence of the characteristic dark-brown, lemon-shaped eggs in the manure permits ready diagnosis.

**Treatment and control.**—No treatment or specific control measures can be recommended. Some of the parasites are removed by phenothiazine.

### THREAD LUNGWORM

**Location.**—The thread lungworm inhabits the air passages of the lungs, the bronchial tubes, and the windpipe.

**Appearance.**—This is a rather long worm, easily observed (fig. 11, *A*). It is white, and the intestine shows as a dark hairline throughout the length of the worm. The male is about 1¼ to 3 inches long; the female, which tapers toward both ends, is 2 to 4 inches long, with a straight, cone-shaped tail.

**Life history.**—Direct. The eggs, each containing an embryo, hatch in the sheep's lungs or digestive tract; both eggs and larvae are coughed up and swallowed. The young larvae pass out with the manure and transform on pastures into infective larvae in about 6 days, under favorable conditions. Infective larvae swallowed by a sheep pass through the intestinal wall to the nearby lymph nodes. After the young worms have been carried from these nodes through the heart to the lungs, they break out of the capillaries and reach the fine bronchial tubes. There they develop into adult worms, which usually become sexually mature by the end of the fourth week after infection.

**Distribution.**—This lungworm is widely distributed. Infestations are rather common, especially where there is plenty of moisture and warmth.

**Damage and symptoms.**—The worms and their eggs and larvae irritate the lining of the air spaces and small tubes in the lungs, causing inflammation and a catarrhal condition. A frothy mucus is produced, sometimes containing traces of blood. The worms or this mucus may block the air passages and cause collapse of some air spaces. This may cause large parts of the lungs to become inactive. Localized pneumonia may result from spread of the inflammation to the lung tissue. Occasionally bacterial infection of the weakened lung tissue may follow; then pus may form and parts of the lungs may become dense.

Often there is an early diarrhea, but ordinarily the symptom first noted is a husky cough. If large parts of the lungs are affected, breathing may become noticeably difficult. Affected animals are likely to die of weakness or suffocation. An experienced veterinarian or a stockman who is familiar with the disease can diagnose it by observing the animal's behavior, but his diagnosis should be confirmed by a laboratory ex-

amination of the manure, or of saliva from the back of the tongue. Occasionally, moreover, such specimens from an infected animal may not contain larvae, especially if the infection is recent. On post mortem, the lungs show inflamed patches and the worms can be found in the air passages. Small accumulations of cheesy pus, sometimes containing worm remnants, may be found at the tips of the small bronchial tubes.

**Treatment and control.**—No completely satisfactory treatment has been found for removing or destroying the thread lungworm. Many kinds of medicine have been tested extensively, by injecting them into the windpipe, but none of them have proved to be sufficiently safe or effective for general use. Also, injecting substances directly into the windpipe and bronchial tubes is dangerous. Control depends chiefly upon proper pasturage and feeding. Avoid pasturing sheep on low-lying, wet areas; keep infected animals in dry lots to avoid constant reinfection. Apply sanitary measures, rotate pastures, and isolate infected animals.

## HAIR LUNGWORM

**Location.**—The hair lungworm inhabits the small bronchial tubes, the air sacs of the lungs, and the lung tissue.

**Appearance.**—This worm is much smaller than the thread lungworm. The male is about ½ to 1 inch long and the female about ¾ inch to 1¼ inches long, but they are very slender and hard to find.

**Life history.**—Indirect. Several different species of land snails and slugs serve as intermediate hosts. The young larvae, carried out in the manure, enter the body of a snail or slug, and there develop into infective larvae. Apparently, infested snails and slugs are swallowed by grazing sheep. This lungworm migrates and develops in the sheep in about the same way as the thread lungworm. Larvae appear in the manure about 6 weeks after infection.

**Distribution.**—This lungworm is widely distributed. It is very prevalent in the Eastern States. In this region, reports indicate, nearly all lambs over 6 months old are infested.

**Damage and symptoms.**—Usually, many of the worms live not far below the outer covering of the lungs. They inflame the lung tissues, and rounded nodules about 1/10 to 4/5 inch in diameter, containing a greenish-gray substance, form around them. As a result many small circular grayish areas, some of them slightly elevated, may mark the surface of infested lungs. Sometimes much of the surface has a grayish or greenish cast. Worms farther from the surface, and accumulations of the worms' eggs and larvae, inflame and damage the lung tissue in a similar way. Sometimes tissues around the nodules become dense.

No symptoms are observed, as a rule, but heavy infestations undoubtedly reduce the amount of functioning lung tissue enough to injure a sheep's general health and resistance.

**Treatment and control.**—In preliminary tests, treatment with emetine hydrochloride has shown promise of being effective against the hair lungworm. A 1-percent solution in water is injected into muscle tissue at a dose rate of 0.15 cubic centimeter per pound of body weight, and this injection is repeated 48 hours later. The treatment should be given only by a veterinarian.

Since a land snail or slug is a necessary link in the life history of this worm, the obvious way to prevent infestation of sheep is to keep them away from places inhabited by snails and slugs—that is, from low, damp areas of pastures. Wherever this trouble appears,

sheep should be pastured on high, dry ground so far as possible. Other general methods of control are the same for the hair lungworm as for the thread lungworm.

### RED LUNGWORM

**Location.**—The red lungworm usually is found in the bronchial tubes.

**Appearance.**—The worms (fig. 11, *G*) are slender and reddish. The males are about ¾ inch to 1½ inches long; the females, a little longer.

**Life history.**—Indirect. So far as known, similar to that of the hair lungworm; however, fewer kinds of land snails can serve as intermediate hosts.

**Distribution.**—In this country, so far, this lungworm has been found in sheep in parts of New York, Virginia, and Wyoming only.

**Damage and symptoms.**—The worms cause local inflammation of the lining of the small bronchial tubes. A fluid exudes from the inflamed areas, and the inflammation spreads to the lung tissue around the affected tubes. Finally, small areas become affected with lobular pneumonia. Infested sheep usually show no definite symptoms, but their general health may be affected and the weakened lungs are more susceptible to bacterial infection.

**Treatment and control.**—The only known control methods are those described for the hair lungworm.

### EYEWORM

**Location.**—The eyeworm is found in the tear ducts, on the surface of the eye beneath the lids (including the third eyelid), and even in the eyeball.

**Appearance.**—The worms (fig. 11, *E*) are slender, whitish, and about ⅛ to ⅕ inch long; usually a very careful examination of the eye is necessary to detect their presence.

**Life history.**—Undetermined; very probably indirect, with some kind of insect acting as the intermediate host.

**Distribution.**—The eyeworm occurs in California only. Thus far, it has not often been found in sheep. It occurs also in dogs, deer, and man.

**Damage and symptoms.**—Eyeworm infestations in sheep have not been given much study thus far; they definitely appear to cause inflammation of the eyeball and inner lining of the eyelids and excessive watering of the eyes.

To detect the worms, it is usually necessary to expose the inner surface of the eyelids, including the third one. They may then be seen wriggling in the tears. Anything that tends to increase the flow of tears, such as application of dressings or use of certain local anesthetics, seems to make it easier to detect the worms.

**Treatment and control.**—No specific methods for treatment and control have been worked out.

### ARTERIAL WORM

**Location.**—The arterial worm lives in the circulatory system. It has been found in the left ventricle of the heart and in the aorta and other arteries.

**Appearance.**—This worm (fig. 11, *J*) is threadlike and whitish. The females are about 4½ to 5 inches long; the males, about 2 inches shorter.

**Life history.**—Undetermined; very probably indirect, with a biting or sucking insect of some kind serving as intermediate host. The female worms produce living larvae, which are carried by the blood to the skin, where they become accessible to insects of these types.

**Distribution.**—The skin condition caused by the larvae of the arterial worm is fairly prevalent in some flocks in New Mexico and Colorado and has been observed on sheep in California and from Ari-

zona, Oregon, and (probably) Utah. Adult arterial worms have been found in sheep in the first three of these States and in deer in California, Arizona, and Utah.

**Damage and symptoms.**—The adult worms, so far as is now known, cause direct injury to sheep only occasionally, but the presence of the larvae in a sheep's skin sets up an inflammation (fig. 17) known as sorehead or filarial dermatosis. This is often limited to the region of the poll, but it sometimes extends forward to the nostrils and lips. The feet and a part of the abdomen may be affected in the same way. The extent of the areas affected seems to depend upon how much the sheep rubs or scratches them. Frequently, the condition is limited to half the face and one of the feet, usually the hind one, on the same side of the body. The lining of the mouth and nasal cavities and the corneas of the eyes sometimes are affected, also. Deformity of the hoofs can result when filarial sores near them become chronically infected.

FIGURE 17.—Sorehead, caused by arterial worm larvae in the skin of the face and poll.

**Treatment and control.**—Several treatments have shown promise. Tartar emetic (potassium antimonyl tartrate) in glucose solution appears to be the most efficient and economical of these. To prepare it, dissolve 1 gram of tartar emetic and 6.4 grams of anhydrous glucose in enough water to make 100 cubic centimeters of solution. A dose of 35 to 40 cubic centimeters is injected into muscle tissue, once a week for 7 or 8 weeks. In general, the reaction of sheep to this treatment has been satisfactory.

Diethylcarbamazine, thiacetarsamide, stibophen, and anthiomaline will have to be tested further before their merits can be fairly evaluated.

## OTHER ROUNDWORMS

Recently, invasion of the brain and spinal cord by adults of a little-known roundworm, *Neurofilaria cornellensis*, was reported as the cause of hindquarter paralysis in a small number of lambs in New York and New Hampshire. This worm probably does not normally live in sheep. Its life history is not known. Recent reports also indicate that invasion of the central nervous system by immature forms of the abdominal roundworm is the cause of a "lumbar paralysis" that periodically affects considerable numbers of sheep and goats in certain Oriental localities. This worm is normally a parasite of cattle and not of sheep. Whether it gets into the nervous system of sheep in the United States is not known; however, the opportunity for it to do so presumably exists, because cattle in this country commonly harbor it.

Sheep in this country sometimes harbor in the digestive tract species of roundworms other than those that have been discussed. Some of these have been found in sheep very infrequently, and none are known to damage them seriously. They are *Gongylonema verrucosum*, which lives in the paunch and is closely

related to the gullet worm; three species of medium, or brown, stomach worms, *Ostertagia occidentalis*, *Marshallagia marshalli*, and *Pseudostertagia bullosa;* two capillarid worms, *Capillaria brevipes* and *C. bovis*, inhabiting the small intestine; the pinworm, which lives in the large intestine and is fairly frequent in goats; and the swine ascarid, which occurs in the fourth stomach and small intestine but does not reach maturity in sheep.

### Flukes

Three species of flukes are known to occur in sheep in the United States often enough to cause serious losses. All three are liver flukes. The common one causes very heavy losses in some large areas of this country. These flukes are unsegmented flatworms shaped something like a leaf. Each adult fluke has both male and female reproductive organs and produces and discharges large numbers of microscopic eggs, which pass out with the infested sheep's manure. Each of the three species has an indirect life history. The intermediate hosts required by two of them, including the common one, are aquatic snails; thus sheep get these two flukes only when kept on areas where ponds, streams, ditches, or swampy lands provide suitable habitats for such snails. The third species develops in land snails and also requires a second intermediate host.

Rumen flukes, species of *Paramphistomum* and *Cotylophoron*, have been found very rarely in sheep in this country.

### COMMON LIVER FLUKE

**Location.**—The common liver fluke is found usually in the canals and ducts of the liver. It occurs also in the gall bladder and, as a wandering parasite, in the lungs and elsewhere.

**Appearance.**—This fluke (fig. 18, *A*) is brownish, flattened, and leaflike. Usually it is about 1 inch long and ½ inch wide, but it may be as much as 2½ inches long and correspondingly wider. The mouth and a sucker are situated on a cone-shaped extension at the head end. Slightly behind this cone is another small sucker.

**Life history.**—The eggs, which pass out in the manure, can develop and hatch on moist soil. When the egg hatches, it releases a free-swimming embryo. If this embryo gets into even a film of water and penetrates into a suitable snail, it gives rise in time to numerous fluke larvae, which leave the snail's body. The intermediate hosts include several species of amphibious or aquatic snails that live on the banks of ponds, streams, and ditches and on marshy or flooded parts of pastures (fig. 19).

Each of the larvae has a tail by means of which it swims about. Finally it loses the tail and, while

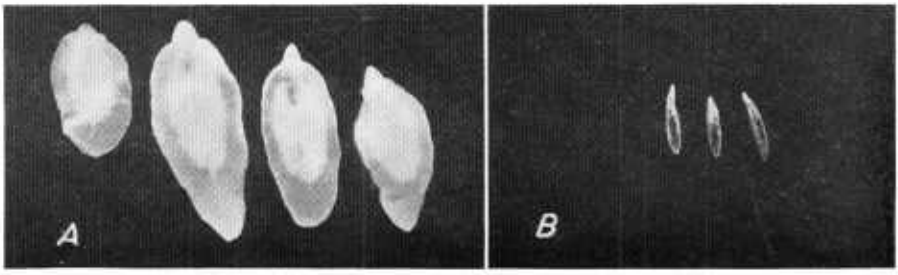


FIGURE 18.—Liver flukes of sheep (natural size): *A*, The common liver fluke; *B*, the lancet fluke.

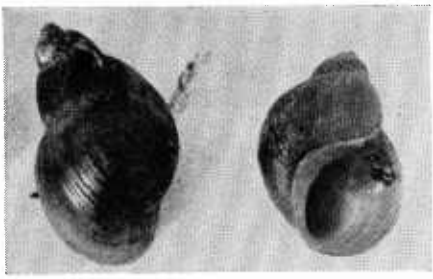

FIGURE 19.—A species of snail, *Lymnaea* [=*Galba*] *bulimoides* Lea, that acts as intermediate host for the common liver fluke. (Enlarged. Courtesy of Oregon Agricultural Experiment Station.)

suspended at the surface of water or after becoming attached to a grass blade or other vegetation, forms a cyst around itself (fig. 20). At this stage the larva is infectious to sheep and some other animals. When it is swallowed with water or forage by a sheep and reaches the intestine, it comes out of its cyst and bores through the intestinal wall to the body cavity. At first the young flukes wander about in the body cavity. As a rule, they then penetrate the liver, live in the liver pulp for a few weeks, and finally enter the smaller bile ducts. A few go astray, perforate the diaphragm, and get into the lungs. The young flukes in the liver make their way down the bile canals until they reach the larger bile ducts, where they develop to sexual maturity. Eggs begin to appear in the sheep's manure about 10 to 12 weeks after infection, and the life cycle begins again.

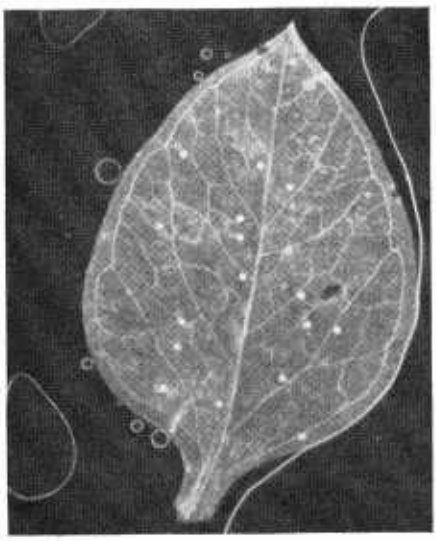

FIGURE 20.—Infective larvae of the common liver fluke (seen as whitish spots) on leaf. (Natural size. Courtesy of Oregon Agricultural Experiment Station.)

**Distribution.**—The known distribution of this fluke includes the Pacific States, the Gulf Coast States (Mississippi excepted), the Mountain Region, and Arkansas. Infestations are common in these areas and are most prevalent in the Pacific States, Utah, Texas, Arkansas, Louisiana, and Florida.

**Damage and symptoms.**—The liver is damaged by the entrance and migrations of the young flukes. It becomes swollen and bleeds into the abdominal cavity, and fluid from it accumulates in this cavity. The presence of the mature flukes causes the bile ducts to become thickened; in advanced cases, the ducts stand out prominently and their walls become hard and gritty.

If the liver is suddenly invaded by a large number of young flukes, the sheep becomes acutely sick and may die after an illness of only a few days. In such a case the symptoms are not ones that would ordinarily be recognized as due to liver flukes.

Sheep that survive heavy infections may show bottle jaw while the young flukes are migrating in the liver. "Potbelly" develops during the early stages of such infections, but it disappears when the flukes reach maturity.

In chronic cases due to lesser infestations, the sheep is likely to put on fat and seemingly improve in condition in the early stages of the disease, usually in the summer and fall. Apparently this seeming

improvement results from a stimulation of the functions of the liver. Later, however, the animal loses condition. The skin and mucous membranes are paler, and the animal is less lively. It feeds less and chews the cud less. As the composition of the blood is altered, a swelling commonly appears under the jaw. "Potbelly," too, often develops. During the winter the sheep becomes leaner, breathes rapidly and feebly, and looks dejected. Diarrhea is usually present at a late stage of the disease.

The animal may die at any stage of the disease. If it survives, some or all of the flukes finally may leave it. In that case part of the damage is repaired. Total recovery is hardly possible, as the liver is burdened with scar tissue in areas where the flukes have been. Although the disease can be diagnosed from symptoms alone by an expert, a safer diagnosis is based on proof that eggs of the fluke are present in the manure. The safest diagnosis is made by killing a sick sheep and finding the flukes in its liver. The ducts of the liver are carefully slit and may be directly examined. If present, the flukes are seen as dark, leaflike objects that, if watched for a short time, can be seen to move. A more satisfactory method is to wash the liver in a plentiful supply of clear water while cutting it up and then promptly and carefully examine the water for flukes. More elaborate steps must be taken to determine whether immature flukes are present in the liver tissue. The liver of an infected sheep may show channels under the membrane that covers it. In old cases, puckered scar areas are present. The bile canals and gall ducts often are marked by ridges on the surface of the liver.

The bacteria that cause black disease, or liver rot, may be present in the livers of healthy sheep. When the liver is damaged by the entrance of the flukes, these bacteria may become active and this fatal disease may develop.

**Treatment and control.**—Two treatments, carbon tetrachloride and hexachloroethane, are available for destruction of the common liver fluke of sheep. Unfortunately, neither of these acts effectively against immature flukes, and neither can be depended on to destroy flukes in a liver that has already been severely damaged. In the dosages used in treating the common liver fluke, both are useful also against the rumen flukes.

Carbon tetrachloride is the preferred treatment, for reasons of economy and simplicity. Adult animals are usually given 2 doses of 1 cubic centimeter each, one shortly after the close of the grazing season and the other about a month later. The standard dose for lambs, given at the same times, is one-half cubic centimeter. The chemical is given either in capsules or as a drench. Usually the drench is prepared by dissolving the chemical in from 2 to 5 parts of mineral oil. For reasons not understood, animals occasionally die from these doses. Experience in Australia suggests that doses twice as large are more efficient against flukes and involve little, if any, added risk to the animals.

Hexachloroethane acts about as effectively against the common liver fluke as carbon tetrachloride. A dose of 30 cubic centimeters (1 ounce) of a hexachloroethane-bentonite-water suspension, containing 15 grams of hexachloroethane, is given to an adult sheep, and one of 15 cubic centimeters (one-half ounce) to a lamb. The safety of the treatment is about the same as that of carbon tetrachloride.

Because all flukes require a snail intermediate host for completion of their life cycles, a principal means of combating them is effective control of snails. This may be brought about by drainage of wet areas where snails propagate, by judicious

use of copper sulfate or other snail-destroying chemicals, or, preferably, by a combination of the two. However, any such chemicals may injure vegetation, livestock, and fish. Application of these materials must be entrusted only to someone thoroughly familiar with the hazards involved.

### LARGE LIVER FLUKE

**Location.**—The large liver fluke almost always occurs in the liver tissue, in a cyst containing, together with one or several flukes, a quantity of dark-colored fluid filled with dead tissue. Occasionally it inhabits the liver ducts or wanders to the lungs or other organs.

**Appearance.**—This fluke (fig. 21) may reach a length of about 4 inches and is much fleshier and broader, or more oval, than the common liver fluke. The front sucker is not carried on a distinct cone, but in its general appearance otherwise the fluke resembles an overgrown specimen of the common liver fluke.

**Life history.**—Essentially the same as that of the common liver fluke; the known intermediate hosts include most of the same snails. However, the larvae do not become encysted at the surface of the water and are said to encyst mainly on coarse vegetation rather than on grass blades. When the young flukes reach bile ducts while moving through the sheep's liver, instead of moving out into the ducts (as young common liver flukes do) they keep on moving through the liver tissue until it reacts and forms cysts around them. They reach sexual maturity about 5 months after infection.

FIGURE 21.—The large liver fluke. (Natural size.)

**Distribution.**—This fluke has been found in sheep in Montana, Idaho, Minnesota, and Wisconsin. It is primarily a parasite of deer, and has been found in them not only in those States but also in Michigan, Texas, Arkansas, Florida, and Louisiana.

**Damage and symptoms.**—Infestations with the large liver fluke may cause severe damage. The symptoms resemble those of infestation with the common liver fluke. Sheep infected experimentally became very thin and weak even though on post mortem examination they were found to have only a few flukes in their livers.

Post mortem examination of the liver reveals the characteristic cysts or else dark-bluish scars where the flukes have formerly been. Apparently the flukes die in the liver. The cysts, which may be present in the lungs and spleen as well as in the liver, take on the character of abscesses. Affected livers and other organs contain black pigment, a characteristic sign of the presence of this parasite. The infestation may cause peritonitis, and the visceral membranes may show black markings.

**Treatment and control.**—No specific medication can be recommended. Draining or filling wet areas or careful use of snail-destroying chemicals is advised.

### LANCET FLUKE

**Location.**—The lancet fluke lives in the bile ducts and the gall bladder.

**Appearance.**—Lancet flukes (fig. 18, *B*) are only about ¼ to ½ inch long and about 1/12 inch wide.

**Life history.**—The life history differs from that of the common liver fluke in several ways. Two intermediate hosts are required, a land snail and a black ant. The eggs that pass out with the manure of an infested sheep do not hatch in the open. When eaten by the snail, they hatch and finally give rise to larvae that pass out of the snail's body in gelatinous "slimeballs." When this material is eaten by the ant, the larvae in it become encysted in the ant's body. When infested ants are swallowed by a sheep, the young flukes are released in the gut. By way of the gut wall and the blood they reach the liver and bile ducts, and there they mature. Eggs appear in the manure about 11 weeks after infection.

**Distribution.**—Although it has been found only in a few flocks in New York, this fluke is expected to become more widely distributed. It occurs in cattle and certain wild animals in central New York, and the intermediate hosts that transmit it in New York are widely distributed elsewhere.

**Damage and symptoms.**—The lancet fluke irritates the bile ducts and causes their walls to thicken. This puts pressure on the liver cells near the ducts and causes them to degenerate. Generalized liver cirrhosis may finally result. The symptoms resemble those caused by the common liver fluke. They appear only in heavily infested animals.

**Treatment and control.**—No specific medicinal treatment can be recommended. General control measures directed against the snail intermediate hosts are advisable. No way is known of controlling the ant intermediate host.

## Adult Tapeworms

Three species of adult tapeworms infest sheep in this country—two broad tapeworms and the fringed tapeworm. They live in the digestive system. Each has a head

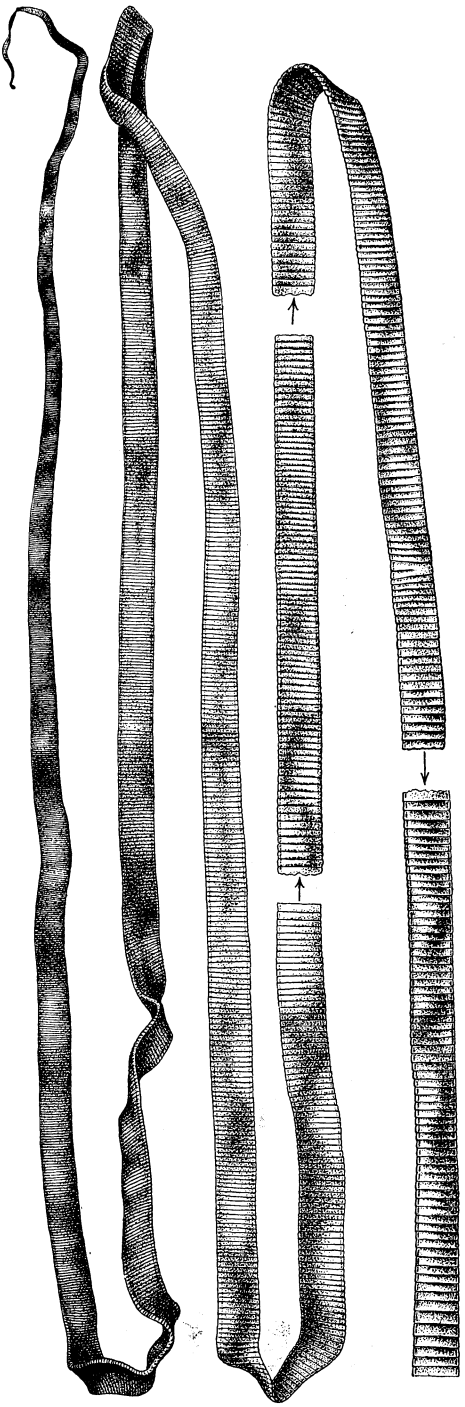

FIGURE 22.—Adult broad tapeworm of the species *Moniezia expansa*. (About natural size.)

bearing suckers and a body composed of flat segments attached somewhat like links of a chain. Both male and female reproductive organs develop in each segment. As the chain lengthens, the oldest segments become filled with eggs and from time to time some of them break off from the body. These then pass out in the manure. The broad tapeworms are known to require intermediate hosts.

## BROAD TAPEWORMS

**Location.**—The broad tapeworms, *Moniezia expansa* and *M. benedeni*, inhabit the small intestine.

**Appearance.**—These worms (fig. 22) are whitish to yellowish and grow to a great length, sometimes several yards. Each segment is broader than long.

**Life history.**—Indirect. The eggs, which pass out in the infested sheep's manure, usually enclosed in segments that soon disintegrate, become scattered on the pasture. Each contains a 6-hooked embryo, which does not develop further while the egg is in the open. When the embryo is swallowed by any of several different species of tiny free-living mites, called beetle mites (fig. 23), which crawl about on the soil and vegetation as they feed on plant debris, it finally develops into a larva in the mite's body. This larva consists mainly of a head enclosed in an almost spherical cyst and is infectious to sheep. When a sheep swallows an infested mite, the larva is liberated into its digestive tract. The head, which bears suckers, comes out of the cyst and passes to the sheep's small intestine. There it becomes attached and segments grow from its base. The worm develops to sexual maturity in the intestine about 35 days after infection.

**Distribution.**—The broad tapeworms are widely distributed and infest sheep rather commonly, *M. expansa* being present much more frequently than *M. benedeni*. The

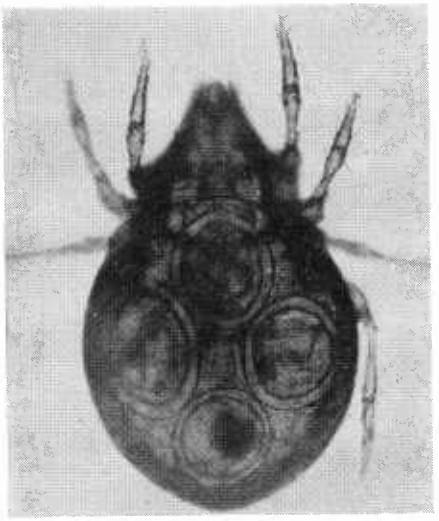

FIGURE 23.—Beetle mite, intermediate host of one of the broad tapeworms, *Moniezia expansa*. The four spherical objects within the body cavity are encysted infective larvae of this species. (Very greatly enlarged.)

actual number of worms in one infested sheep usually is not large, but sometimes their bulk is enough to fill a quart jar.

**Damage and symptoms.**—Some investigators have regarded these tapeworms, *M. expansa* in particular, as serious parasites of sheep, causing marked loss of weight, severe anemia, digestive disturbances manifested by diarrhea, and even death. This opinion is not borne out by the results of experiments. Lambs that were given *M. expansa* infestations have shown no effects or, at most, none more serious than slight digestive disturbances, temporarily resulting in softened droppings, very slight anemia, and slight retardation in growth. However, naturally infested lambs that harbored some roundworms, also, and were scouring badly, stopped scouring and improved in general condition when treatment with a drug had removed their tapeworms. The presence of chains of whitish segments, or of individual segments, in the manure

permits a diagnosis of infestation, provided a reliable identification is made. An individual segment tends to stick to the surface of a pellet and is large enough to be noticed readily.

**Treatment and control.**—Lead arsenate is now the preferred treatment for eliminating broad tapeworms from sheep.[8] Several other treatments are useful. These include some already described in the section dealing with stomach worms—copper sulfate-nicotine sulfate drench (p. 17) and the copper sulfate drench (p. 18).

The acid spray grade of lead arsenate, readily available, is used in doses of 0.5 gram for lambs and 1.0 gram for animals over 60 pounds. It can be given conveniently in small gelatin capsules. If roundworms, as well as tapeworms, are present, the lead arsenate can conveniently be given in combination with phenothiazine used for control of the roundworms. Lead arsenate, in addition to being effective and safe, is economical and easy to use. It must not be given, however, to animals that are to be slaughtered within 4 or 5 weeks.

## FRINGED TAPEWORM

**Location.**—The fringed tapeworm is found, sometimes simultaneously, in the small intestine, the gall ducts, the gall bladder, the fine bile canals of the liver, and the ducts of the pancreas.

**Appearance.**—This worm (fig. 24) is whitish or yellowish. It sometimes reaches a length of 2 feet but usually is not longer than 7 inches. It can readily be distinguished from the broad tapeworms by the fact that each of its segments has a fringe at the rear edge. When the segment is in water, the fringe floats out and can be seen more easily.

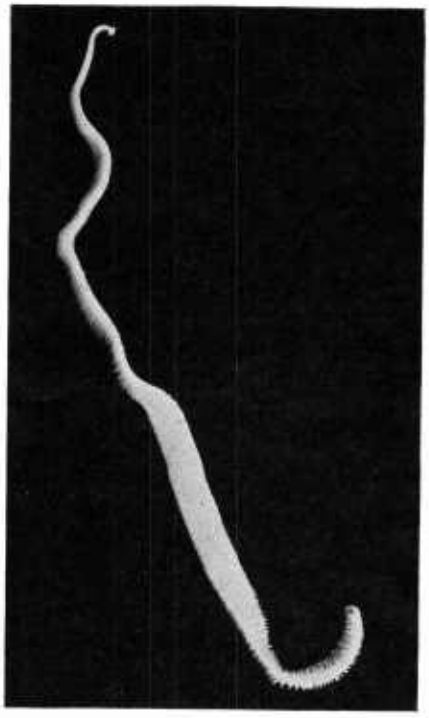

FIGURE 24.—Fringed tapeworm. (Natural size.)

**Life history.**—The life history is not yet known. It is generally assumed to be indirect.

**Distribution.**—This tapeworm occurs primarily in western range sheep; it is found in the East only in sheep that have been shipped from the West. The infected range probably is confined to North Dakota, South Dakota, Nebraska, Kansas, Oklahoma, Texas, and the States west of them. In New Mexico, 60 percent or more of sheep are infested.

**Damage and symptoms.**—The worms usually cause the bile ducts, particularly the common bile duct, to become dilated, thickened, and inflamed. Other changes have been noted in the duct walls, but these may not be caused primarily by the worms. It is doubtful that the ducts ever become completely

[8] Other metallic arsenates—those of iron, copper, and calcium—have been found to be as effective as lead arsenate in limited trials; they require further study before specific recommendations can be made.

stopped up. Jaundice of the eye membranes and of the skin tissues is sometimes noted in infested sheep, but it is not known whether this is actually caused by the worms. This tapeworm has been thought to cause unthriftiness, diarrhea, and even death. At present, most investigators consider it of little importance as a cause of disease. However, livers infested with it are condemned for sale as meat, and this causes considerable economic loss to sheepmen.

**Treatment and control.**—No control measures can be suggested other than general sanitary procedures. Dichlorophen, a preparation containing bis (2-hydroxy-5-chlorophenyl)-methane, has shown some action against this tapeworm, but not enough to justify the cost of the treatment.

## Larval Tapeworms (Bladderworms)

Four species of bladderworms infest sheep in this country. They are the larval forms of adult tapeworms that live in the intestines of dogs and of wild animals such as foxes.

### THIN-NECKED BLADDERWORM

**Location.**—The thin-necked bladderworm is found attached to the membranes of the viscera of the abdominal cavity or in the liver.

**Appearance.**—The parasite looks like a sac full of a clear fluid, with a white object, which is the head and neck, projecting into it at one end. Its diameter is usually about 1 inch but may be several inches. A reaction of the tissues of the host animal causes a cyst (fig. 25) to develop around the bladderworm. If this cyst is broken the thin-walled parasite usually rolls out. By careful use of pressure the head and the rather long neck can be squeezed out at one end of the "bladder."

**Life history.**—When one of these bladderworms is swallowed by a dog, the cyst wall is digested, but the head and neck pass on to the small intestine. The head becomes attached to the intestinal wall, and segments begin to grow back from the neck. The worm continues to grow in this manner until it becomes one of the largest of the adult tapeworms of dogs. It matures and begins to shed egg-bearing segments in about 10 or 12 weeks. It reaches a length of a yard or more. When dogs infested with it run over pastures used by sheep they leave droppings containing its eggs. Rain washes the eggs onto the grass and into streams and puddles where the sheep drink. When a sheep takes in these eggs with food or water the embryo escapes from its surrounding shell, bores into the wall of the intestine, gets into the blood stream, is carried to the liver, and there begins to develop. It bores to the surface of the liver. In time it slips out of the liver and becomes attached to a membrane of the viscera of the abdominal cavity. At first it is a bladder without a head, but within about 2 months it develops a head and neck and becomes a fully formed bladder-

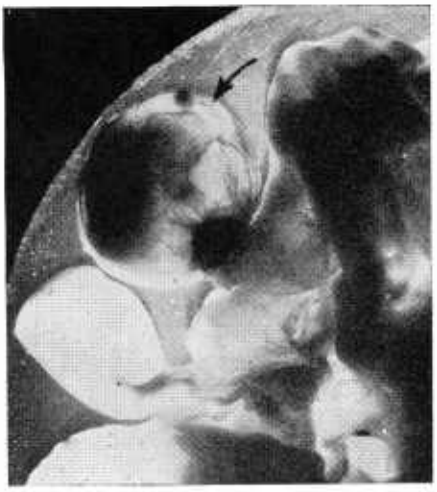

FIGURE 25.—Thin-necked bladderworm (cyst is shown by arrow) attached to a membrane of a sheep's abdominal cavity. (About natural size.)

worm ready to infect any dog that eats it. Some meat-eating wild animals, also, are susceptible to the adult tapeworm.

**Distribution.**—This parasite occurs in most parts of the United States, but apparently it is becoming less common as a result of improvements in disposal of offal at slaughterhouses. It is most likely to be present in sheep that are associated with dogs in vicinities where sheep, hogs, or cattle are slaughtered on farms or at small country slaughterhouses and little care is exercised in disposing of viscera and diseased parts of carcasses.

**Damage and symptoms.**—Light infestation seems to do a sheep little damage. A severe infestation, resulting from the swallowing of an entire segment full of eggs, makes the animal very sick and may kill it. If death occurs, it usually comes at an early stage of the disease, while the embryos are wandering around in the liver. Early death may be preceded by hardly any symptoms, although usually it is preceded by depression, weakness, and lack of appetite. The immediate cause of early death is hemorrhage from the liver or else peritonitis. In such a case, the parasites are so small that they are likely to be overlooked in post mortem examination. However, a liver that has just recently been invaded by large numbers of the parasites usually has ridges or wavy markings on its surface and may have burrows in its substance, which show the course of the wanderings of the young worms.

On post mortem examination of an animal that has been infested for a longer time, it is usually easy to see the bladderworms in the membranes of the viscera of the abdominal cavity or, in earlier stages, in the liver.

**Treatment and control.**—There is no medicinal treatment for bladderworm infestations, and surgical treatment can rarely be given. The recommended control methods are preventive: Proper disposal of infected sheep carcasses or parts of carcasses, elimination of predatory or stray dogs and other meat-eating animals, and examination and treatment, with appropriate remedies, of any dogs that come in contact with sheep.

### SHEEP MEASLES WORM

**Location.**—The sheep measles worm occurs most commonly in the heart or diaphragm. It tends to invade also the muscles used in chewing and the tongue. It is sometimes found in other muscles and in connective tissues between muscles. Dead larvae sometimes are found in the lungs, the walls of the first and fourth stomachs, and the kidneys.

**Appearance.**—This oval bladderworm occurs in cysts one-seventh to one-third inch long and one-twelfth to one-sixth inch wide. Removed from the cyst, it appears as a thin-walled membranous sac containing a clear fluid and enclosing, about midway between its ends, a tiny, opaque white object, the head and neck (fig. 26). Cysts in which the larvae have died appear as nodules containing a cheesy or, if lime salts have been deposited in them, a hard, gritty substance.

**Life history.**—Similar to that of the thin-necked bladderworm. However, on reaching the sheep's liver the embryos do not bore to its surface, but enter the liver veins and are carried to the heart. From there the general circulation carries them to muscles in which they become fully formed bladderworms in about 2½ to 3 months. So far as known, the dog is the only host of the adult tapeworm that develops from this bladderworm.

**Distribution.**—The sheep measles worm seems to be most common in the West, especially in Montana, Idaho, Washington, Oregon, Cali-

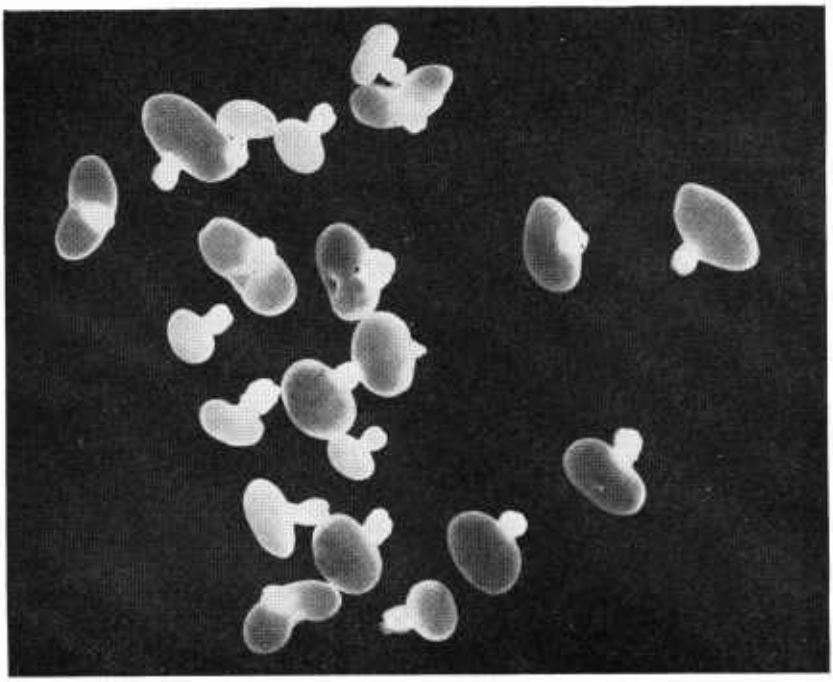

FIGURE 26.—Bladderworms of the species that causes sheep measles, some with head and neck protruding. (About twice natural size.)

fornia, Colorado, and Nevada. Infestations apparently are becoming less frequent.

**Damage and symptoms.**—If a sheep swallows a large number of the eggs within a short time, it may become very sick. One that picks up an entire segment filled with eggs is likely to die about 2 or 3 weeks later. When sheep have only a few measles worms, no symptoms are likely to be observed. On post mortem examination the cysts are the principal abnormality observed. The meat of heavily infested sheep may be watery and discolored.

This parasite is important chiefly because it affects the usability of the meat. Heavily infested carcasses are condemned for food purposes and used only for products not intended to be eaten. If the infestation is light, the infested parts of the carcass are condemned and the remainder is passed for food.

**Treatment and control.**—Recommendations are the same as for the thin-necked bladderworm.

## GID PARASITE

**Location.**—The gid parasite occurs in the brain or spinal cord. Larvae that fail to reach the central nervous system may lodge in muscles and other tissues, where they degenerate.

**Appearance.**—This parasite is often as big as a hen's egg or larger. It appears as a transparent bladder containing a rather large amount of clear fluid and, attached to the bladder wall, a number of white objects, about the size of a grain of wheat, which usually project into the fluid (fig. 27). Each of these white objects is a head.

**Life history.**—Essentially similar to that of the thin-necked bladderworm (p. 41); however, when a dog, fox, or coyote eats a single gid parasite, each of the parasite's

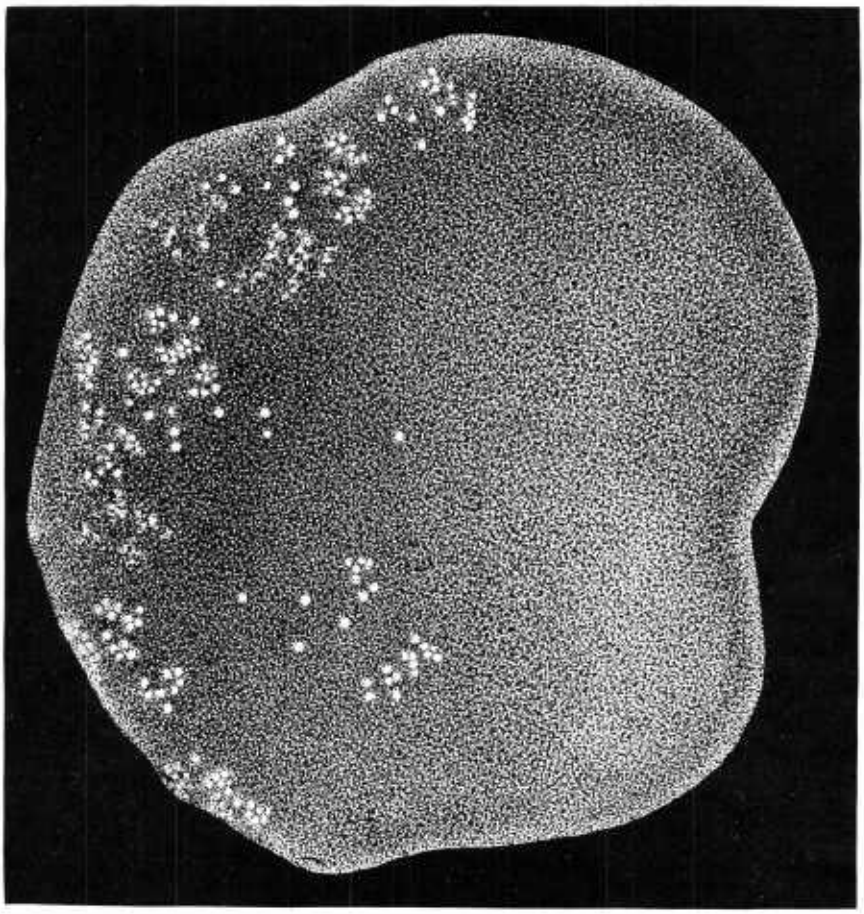

FIGURE 27.—Gid parasite from brain of sheep. (Natural size.)

several heads can attach itself to the animal's small intestine and there develop into a specimen of the adult tapeworm. The sexually mature adult specimen is about a foot long. Also, when a sheep swallows eggs of this worm, the embryos that get into its blood do not bore into the liver substance. They are carried by the blood to various parts of the body. Those that reach the brain or spinal cord wander around in it for a time before they settle down and begin to grow, but they develop into fully formed bladderworms in about 7 to 8 months. The ones carried elsewhere degenerate and die before they grow beyond the size of a pea.

**Distribution.**—In the United States this parasite has been found chiefly in northern Montana, where it has been established since about 1890 or longer. There have been outbreaks of gid in Arizona and in New York. When the parasite has been found in sheep in States other than Montana, the sheep have generally been traced to Montana or to a foreign country.

**Damage and symptoms.**—The channeling movements of the young worms in the brain cause inflammation of the brain tissues. If only a few parasites are invading the brain, about 2 to 3 weeks after infection there usually are some symptoms—slight fever and restlessness—that

can easily be overlooked. Some sheep so infested show more noticeable signs—dullness, imperfect eyesight, and dizziness—for a period of about a week beginning at this time. If a sheep has become heavily infested and has 10 or more young worms migrating in its brain, it looks depressed and sleepy about 2 weeks after infection. It soon loses its appetite and rapidly loses flesh (fig. 28). Its eyes become reddened and its eyesight is markedly affected. Some animals become completely blind and run into objects in their path. Disturbances unrelated to blindness appear at about the same time. The head may be held outstretched or in some other peculiar position. The animal stumbles, falls down frequently, settles on its hindquarters or forequarters, and may lie down for whole days as if paralyzed. Nearly all animals so affected die of brain fever, about a week to a month after the first symptoms appear. Examination then shows a number of curving channels on the surface of the brain. But the parasites are hard to find if no more than about a month old, because they are then only about as big as flaxseed.

The parasites stop channeling through the brain about a month after infection, and for the next 4 to 6 months no particular symptoms are noted, as a rule, even in animals that have earlier shown symptoms of severe infestation. Then, about 5 to 7 months after infection, when the bladderworms have become fully developed, the peculiar behavior known as gid or as turning, or circling, disease begins. It evidently results from pressure and irritation caused by the cysts. Disturbances in both movement and eyesight are noted. Some animals walk in circles—either to the right or to the left—or pivot in one spot, and others walk with the head high and with high knee action, or with the head low and a stumbling gait, the nature of the movements depending on the location of the cysts in the brain. The animals refuse feed and water and become very thin. They may either move

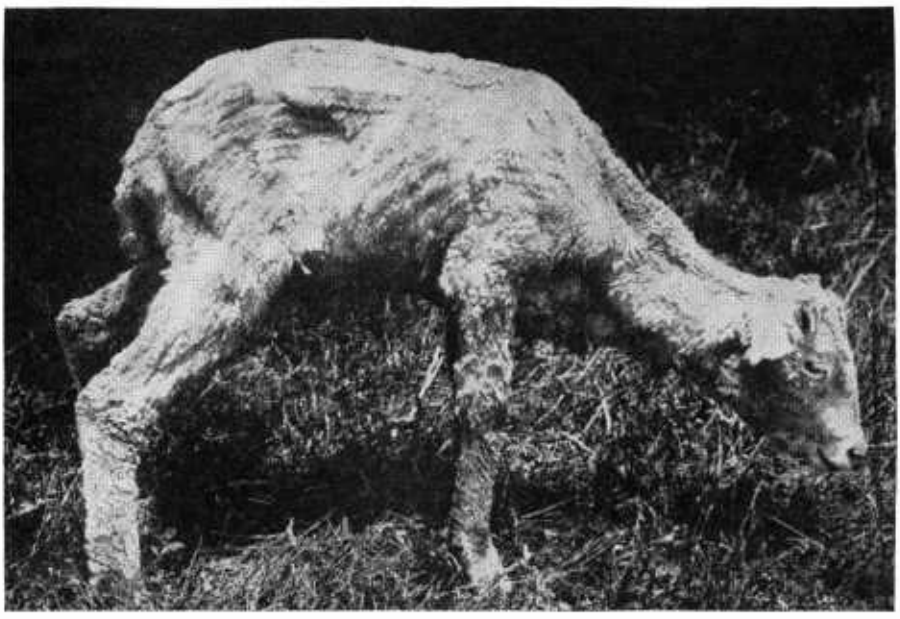

FIGURE 28.—A "giddy" sheep.

about continuously or stop and gaze fixedly into space. They are difficult or impossible to herd, and tend to lag behind the flock or become lost. If the parasite is in the spinal cord, there is paralysis of the hindquarters, the rectum, and the bladder. At first the hind legs are brought up convulsively; later they drag, and their muscles waste away. Death usually follows.

When examined after death the brain or spinal cord is found to have on or in it one or more bladderworms. Some of the brain or cord tissue has disappeared or been crowded aside by the growth of the cyst. Often the part of the skull next to the parasite has been softened or has even been absorbed to such an extent that it has a hole or several holes through it. Sheep in the late stages of the disease are so thin that the meat is unfit for food.

**Treatment and control.**—The only measures that can be used are those described for the thin-necked bladderworm.

### HYDATID WORM

**Location.**—The preferred sites of the hydatid worm in sheep are the liver and lungs, but it may occur in practically any organ or tissue.

**Appearance.**—This worm occurs within cysts formed by the host tissue, often about as big as a walnut but varying from the size of a pea to that of a large grapefruit, sometimes almost a sphere and sometimes irregular in shape. If located on the surface of the organ, the cysts appear as protuberances (fig. 29). The worm itself resembles a transparent bladder filled with clear fluid. The bladder wall, though many-layered, is thinner than the cyst wall. The bladderworm may be single, but usually one gives rise to others that develop inside or outside it and attached or unattached to it. The bladderworm may be sterile, but is

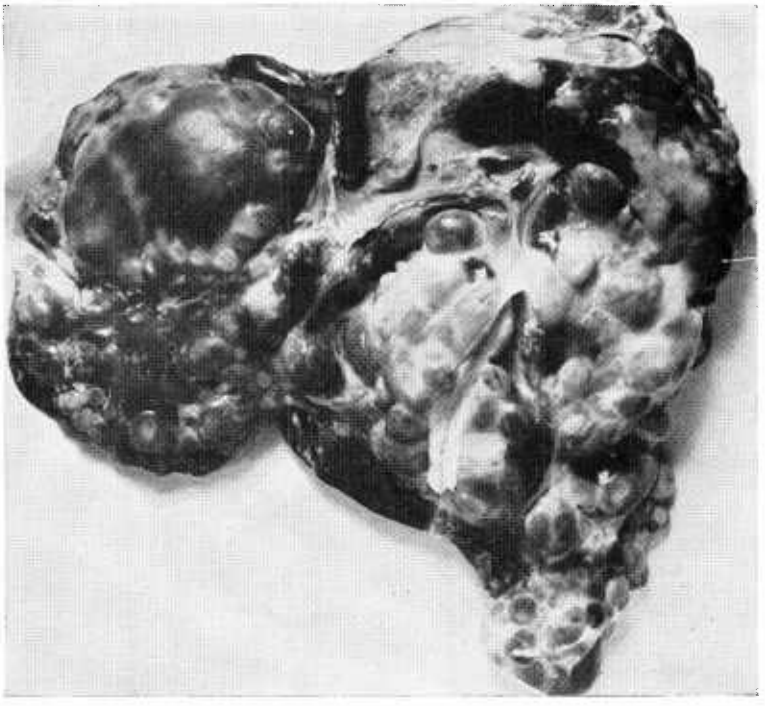

FIGURE 29.—Sheep liver showing hydatid cysts. (Reduced.)

usually fertile. If it is sterile, the bladder contains fluid only; if it is fertile, the bladder contains also tiny objects resembling grains of sand attached to the bladder wall or lying unattached in the fluid. These are brood capsules, and each of them contains several very small heads.

**Life history.**—Essentially like that of the thin-necked bladderworm (p. 41); however, when a fertile hydatid is eaten by a dog or a susceptible wild meat-eating animal each head in each brood capsule it contains can attach itself to the animal's small intestine and there develop into a specimen of the adult tapeworm. The sexually mature worm is only about 1/5 inch long and has only a few segments. The embryos that emerge from its eggs when these are swallowed by a sheep get from the sheep's intestine into its blood stream and may be carried to almost all parts of its body. Usually, however, they become localized in the lungs or liver and there a miniature cyst is formed around each of them in about 10 days. The development of a fully formed fertile hydatid worm takes about 5 to 6 months.

**Distribution.**—Meat-inspection records on liver condemnations show that the hydatid worm is rather widely distributed through the United States. Exact figures on its occurrence in sheep are not available. Contrary to experience in foreign countries, it is found here much less commonly in sheep than in swine or cattle. In the period 1943–52, on an average, United States meat inspectors condemned about 3,000 cattle livers for hydatid infestation each year.

**Damage and symptoms.**—The symptoms vary widely according to the location of the parasite and its size. If the parasite is small or has room to develop without crowding important organs, few symptoms may be noticed. However, hydatid worms in the brain or heart may cause very marked symptoms or even sudden death from pressure or rupture. As a rule, infestations are unlikely to be correctly diagnosed during life. On post mortem examination, large hydatids are readily found.

**Treatment and control.**—The only measures that can be used are those described for the thin-necked bladderworm. Hydatid disease has an important public-health aspect because man, as well as sheep, swine, and related animals, may become infested with the hydatid worm if he accidentally swallows its eggs. Dogs that are suspected of harboring this tapeworm should be handled very cautiously, to prevent human infection, until they are effectively treated with arecoline hydrobromide or else destroyed.

## Protozoa

Protozoa are usually described as one-celled animals. Most of them are much too small to be seen with the unaided eye. Several different kinds of protozoan and protozoan-like parasites occur in sheep in this country. The most important of these are the coccidia, which are true protozoa. They grow and multiply in the wall of the digestive tract, forming microscopic bodies called oocysts, which pass out with the manure of the host sheep.

### COCCIDIA

**Location.**—*Eimeria arloingi*, *E. ninae-kohl-yakimovae*, and *E. faurei*, 3 of the 9 species of coccidia that have been reported from sheep in this country, have been found to occur in cells of the surface layer (epithelium) of the mucous membrane that lines the small intestine; the first two species also inhabit cells of a more deeply situated layer of this membrane. The layer in which they occur depends upon the stage they have reached in their development. *E. ninae-kohl-yakimovae* invades the lining of the

large intestine, also. The six additional species, likewise of the genus *Eimeria*, presumably occur in the mucous membranes of the digestive tract, but the exact locations of the cells they inhabit are not known.

**Appearance.**—Coccidia are not visible to the naked eye. Under a microscope they can be seen as cigar-shaped, spherical, or egg-shaped bodies in scrapings from the lining of the intestine of an infested sheep.

**Life history.**—Direct. The oocysts roughly correspond to the eggs of worm parasites. According to species, they are more or less spherical or football-shaped and are of different sizes. Under favorable conditions they develop to an infective stage on pastures, and elsewhere in the open, in the course of a few days. When an infectious oocyst is swallowed by a sheep, minute cigar-shaped bodies (sporozoites) are liberated from it into the digestive tract.

Each sporozoite of the species *E. arloingi* moves to and enters a cell situated fairly deeply in the mucous membrane of the small intestine. There it grows, undergoes a transformation, and finally gives rise to a numerous brood of somewhat similar bodies called merozoites. When the host cell breaks down, the merozoites are liberated and each of them invades a neighboring cell.

Some merozoites develop into ovumlike bodies, others give rise to spermlike bodies. When these two types of bodies fuse, oocysts are formed and the cycle of development in the host comes to an end. The oocysts are developed in the innermost lining of the intestine. They pass into the cavity of the intestine when the cells harboring them break down. The first ones formed pass out with the manure about 20 days after infection.

**Distribution.**—Several of the species are widely distributed. It is highly probable that scarcely a sheep escapes infection by one of the common coccidia, and most sheep are infected with more than one.

**Damage and symptoms.**—Coccidia destroy the intestinal cells they invade and sometimes the neighboring cells, also. Some of the species may cause much of the intestinal lining to slough off, and at least one species, *E. ninae-kohl-yakimovae*, causes hemorrhages in heavily infested intestines.

Softened droppings may be the only noticeable symptom of light coccidial infections. Heavy infections can cause a serious disease called coccidiosis. Its most striking manifestation is a copious diarrhea. Severely affected lambs and sheep become dull and lose their appetites. The diarrhea may continue for several days or for weeks, causing weakness, loss of flesh, and death. Flecks of intestinal mucous membrane and variable amounts of a bloody discharge may appear in the droppings. At autopsy, inflammation may be found in both the small and the large intestine.

In a coccidiosis experiment conducted during the warm season, the soiled hindquarters of the scouring animals attracted flies and some of the animals became infested with maggots and died, although they probably would not have died from their coccidial infections alone. Marked loss of wool occurred about 1 to 2 months after severe coccidiosis was produced by inoculations with the species *E. faurei* and *E. ninae-kohl-yakimovae*.

Since under natural conditions sheep usually harbor a mixture of coccidial species, coccidiosis as seen in the field is ordinarily a complex of the effects of two or more species. Its severity may depend upon what particular species are present; it is known that the species are not all equally harmful. The effects of the species *E. arloingi*, *E. faurei*, and *E. ninae-kohl-yakimovae* have been

tested separately. Each of these species caused severe diarrhea and related disabilities when large enough numbers of its oocysts were given to lambs. Inoculation with oocysts of the species *E. ninae-kohl-yakimovae* also caused deaths, but there were no deaths among lambs infected with much larger numbers of oocysts of either of the two other species. More blood and blood-tinged mucus were found in the droppings of the lambs infected with the species that caused deaths.

Little is known as to the importance of coccidial infections in farm flocks. Apparently because nearly all adult sheep are carriers of coccidia, lambs kept with their dams and reared in a normal way ordinarily become infected while very young. However, the lambs do not seem as a rule to be made noticeably sick by these infections, particularly if they are well nourished and thrifty. They may scour for a short time, particularly when 6 weeks to 2 months of age. Ordinarily they stop scouring and do not seem to be affected to any marked extent as they grow older, although they continue to be exposed to infection and to harbor coccidia. It is likely, however, that these early infections may retard their growth to a greater extent than is realized.

So far as reports indicate, severe coccidiosis occurs mainly among lambs in feed lots. Typically, widespread scouring begins, and some deaths occur, as early as 2 weeks after the lambs are put in the lots. Death losses mount and may continue at a high level for 2 or 3 weeks, after which they may gradually subside. Many of the lambs that survive may continue to scour and do badly for several weeks longer.

**Treatment and control.**—No specific treatment can now be recommended to the sheepman, although many have proved promising in limited experiments and field tests. These include elemental sulfur and certain sulfonamides, namely, sulfaguanidine, sulfamethazine, phthalylsulfathiazole, succinylsulfathiazole, and sulfadiazine.

Sulfur has no value as a remedy for coccidiosis, but it has been used successfully to prevent the disease. For this purpose, 1 to 1.5 percent of flowers of sulfur is incorporated into the ration for continuous feeding. This method is particularly well suited for use in crowded feed lots. The sulfonamides have more preventive than curative value in relation to coccidiosis. However, no reliable or economical method has been devised for using them as preventives. In treating coccidiosis the sulfonamides named above are usually given for about a week to 6-month-old lambs, in daily doses of 1 to 3 grams, or 0.25 to 1.0 gram for each 20 to 25 pounds of body weight.

Outbreaks of fatal coccidiosis among feeder lambs have been associated rather definitely with the way the animals had been managed. These outbreaks have been traced principally to starvation and exposure caused by poor shipping conditions, improper feeding when the lambs first arrived at the feed lots, and failure to keep the feed troughs clean. Investigation of a typical outbreak of feed-lot coccidiosis showed that practically all the lambs harbored coccidia when they were brought in to the feed lots from farms or ranges. Their manure, containing oocysts, got into the feed troughs. The practice was to add fresh silage twice daily to the uneaten remainder of previous feedings without cleaning out the troughs. Unconsumed old feed gradually accumulated in the troughs beneath the fresh additions, and the manure became scattered through it. In the manure mixed with the moist silage the oocysts had ideal conditions and developed to the infective stage in large numbers. As a result, the lambs eating from the troughs were suddenly

heavily exposed to infection and came down with coccidiosis.

Efforts to prevent the disease should begin before the animals leave the range. For this purpose the grower should buy only good-quality, well-developed lambs; make sure that they are handled carefully on the way from the range to the feed lot; start them on grass or roughage, and gradually shift to a concentrated feeder ration in which 1 to 1.5 percent of flowers of sulfur is incorporated; and clean out the feed troughs frequently.

## SCIENTIFIC NAMES OF PARASITES

Scientific names of parasites that are referred to in the text of this bulletin by their common names only are as follows:

| *Common name* | *Scientific name* |
|---|---|
| Abdominal worm | *Setaria cervi.* |
| Arterial worm | *Elaeophora schneideri.* |
| Biting louse | *Bovicola ovis* [=*Trichodectes sphaerocephalus*]. |
| Common liver fluke [1] | *Fasciola hepatica.* |
| Common nodular worm | *Oesophagostomum columbianum.* |
| Common scab mite | *Psoroptes equi* var. *ovis.* |
| Eyeworm | *Thelazia californiensis.* |
| Fleeceworm fly | *Phormia regina.* |
| Follicular-mange mite | *Demodex canis* var. *ovis.* |
| Foot louse | *Linognathus pedalis.* |
| Foot-scab mite | *Chorioptes ovis.* |
| Fringed tapeworm | *Thysanosoma actinioides.* |
| Gid parasite | *Coenurus cerebralis* [=*Multiceps multiceps*]. |
| Gullet worm | *Gongylonema pulchrum.* |
| Hair lungworm | *Muellerius capillaris* [=*M. minutissimus*]. |
| Head grub | *Oestrus ovis.* |
| Head-mange mite | *Sarcoptes scabei* var. *ovis.* |
| Hookworm | *Bunostomum trigonocephalum.* |
| Hydatid worm | *Echinococcus granulosus.* |
| Lancet fluke | *Dicrocoelium dendriticum.* |
| Large liver fluke | *Fascioloides magna.* |
| Large-mouthed bowel worm | *Chabertia ovina.* |
| Large stomach worm [1] | *Haemonchus contortus.* |
| Lesser nodular worm | *Oesophagostomum venulosum.* |
| Pinworm | *Skrjabinema ovis.* |
| Red lungworm | *Protostrongylus rufescens.* |
| Screw-worm fly | *Callitroga hominivorax* [=*Cochliomyia americana*]. |
| Secondary screw-worm fly | *Callitroga macellaria* [=*Cochliomyia macellaria*]. |
| Sheep-itch mite | *Psorergates ovis.* |
| Sheep measles worm | *Cysticercus ovis* [=*Taenia ovis*]. |
| Sheep tick | *Melophagus ovinus.* |
| Spinose ear tick | *Otobius megnini* [=*Ornithodorus megnini*]. |
| Stomach hairworm | *Trichostrongylus axei.* |
| Sucking body louse | *Linognathus ovillus.* |
| Swine ascarid | *Ascaris suum.* |
| Thin-necked bladderworm | *Cysticercus tenuicollis* [=*Taenia hydatigena*]. |
| Thread lungworm | *Dictyocaulus filaria.* |
| Threadworm | *Strongyloides papillosus.* |

[1] This common name is applied by some investigators not only to the species whose scientific name is given opposite it here but also to one or more closely related species.



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